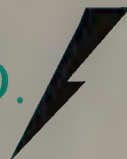


Edition 5

# LOW BACK PAIN SYNDROME



RENE CAILLIET, M.D.



PAIN SERIES



# LOW BACK PAIN SYNDROME

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# **LOW BACK PAIN SYNDROME**

## **EDITION 5**

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# Preface

Thirty years ago the first edition of *Low Back Pain Syndrome* emerged. Over the years voluminous literature has appeared in numerous journals and texts from many countries. Clinical studies have ensued along with essential basic scientific studies in anatomic, biologic, and engineering aspects of low back pain.

Unfortunately, a thorough review of all the above has resulted in the subject remaining obscure, the cause an enigma, the sequelae ominous, and the treatment ineffectual. The mechanical aspects of the lumbosacral spine have been well documented. The ensuing pain is being thoroughly researched and implemented. The financial burden placed on the patient and health-care industry continues to be documented and lamented.

The first edition and subsequent editions of *Low Back Pain Syndrome* stressed the aspects of functional anatomy with its clinical manifestations and evaluation. Clinical experience during the preparation of these volumes revealed that a truly meaningful examination has never been proposed, the meaning of the findings has remained obscure, and the treatment protocols have provided ineffectual results.

In the original texts the relationship of *impairment* as related to *disability* was not evaluated. The psychological aspects as related to *functional*, *structural*, and *mechanical* aspects of low back pain were not adequately clarified. The significance of radiologic studies as related to symptomatology remained esoteric.

This volume has been the result of soul searching as to the true meaning of low back pain; its true mechanism; appropriate meaningful treatment; and proper association of the psychologic, hormonal, as well as mechanical basis of this painful entity.

Merely to differentiate low back pain with and without leg pain has merit but once so discriminated and categorized the advancement of appropriate examination and treatment must be addressed.

The medical entities involved in evaluation and management of the

patient with low back pain remain confused, misinformed, and misguided. Careful and intensive review of the literature remains unsimplified and difficult to assimilate in a meaningful clarification. Modalities remain placebo in type; physical therapeutic measures remain nonspecific and possibly pleasing yet ineffectual. Much surgery could be eliminated. Chronic pain could be avoided if acute pain and its rationale were understood.

The history taken from a patient reveals the mechanism and the psychodynamics leading to the resultant injury. The precise tissue that is nociceptive, invoking pain, can be ascertained but the aspect of the examination delineating this tissue remains unclear.

This volume is not intended to be a new edition of *Low Back Pain Syndrome* because much, if not most, of the content remains pertinent. This volume is intended to clarify recent findings and refute many outdated and even untrue considerations of low back pain.

Hopefully with these efforts better understanding and management of the patient with low back pain will result. The economic benefits will be welcomed in today's exorbitant cost of personal injury and industrial compensation.

RENE CAILLIET, MD

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## CHAPTER 1

# Structure and Function of the Lumbosacral Spine

To fully understand the symptom of low back pain and evaluate the examination of the lumbosacral spine, one must understand the normal structure and neuromusculoskeletal kinetics of the spine. There is voluminous literature on this subject, which will be summarized in this chapter.<sup>1,2</sup> The neuromusculoskeletal kinetics involving the psychophysiological aspects will also be fully evaluated.

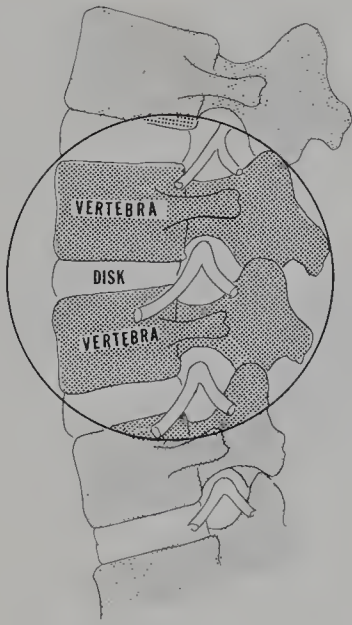
The spine is composed of superincumbent functional units (Fig. 1-1). The lumbosacral spine is usually the lower five vertebrae on the sacrum (Fig. 1-2). Each functional unit is composed of two adjacent vertebrae separated by an intervertebral disk (Fig. 1-3).

The intervertebral disk is a hydrodynamic elastic structure interposed between two adjacent vertebrae (Fig. 1-3) composed of a mucopolysaccharide gel containing annular fibers that attach to adjacent vertebral endplates and obliquely cross each other in planes. The disk has a central nucleus enclosed within annular fiber layers that maintain the separation of the vertebral endplates and allow restricted motion between the adjacent vertebrae.

The nucleus (pulposus) is a proteoglycan substance that contains a random aligned network of type II collagen fibrils (Fig. 1-4). The matrix is hydrophilic, gaining water via an imbibitory mechanism and osmosis.

The surrounding annulus fibrosus comprises 10 to 20 concentric lamellae of collagen fibers (Fig. 1-5).<sup>3</sup> The fibers are essentially oriented at a 65° angle to the longitudinal axis of the vertebral column.

The angulation of the fibers varies as each lamella goes in an opposite direction (see Fig. 1-6). This differential direction has functional



FUNCTIONAL UNIT  
TWO VERTEBRAE  
AND ONE DISK

Figure 1-1. Functional units of the spine. The basic structural component of the spine is termed the *functional unit*. Each unit comprises two adjacent vertebrae separated by an intervertebral disk. (Modified from Cailliet, R: Understand Your Backache. FA Davis, Philadelphia, 1984, p 7.)

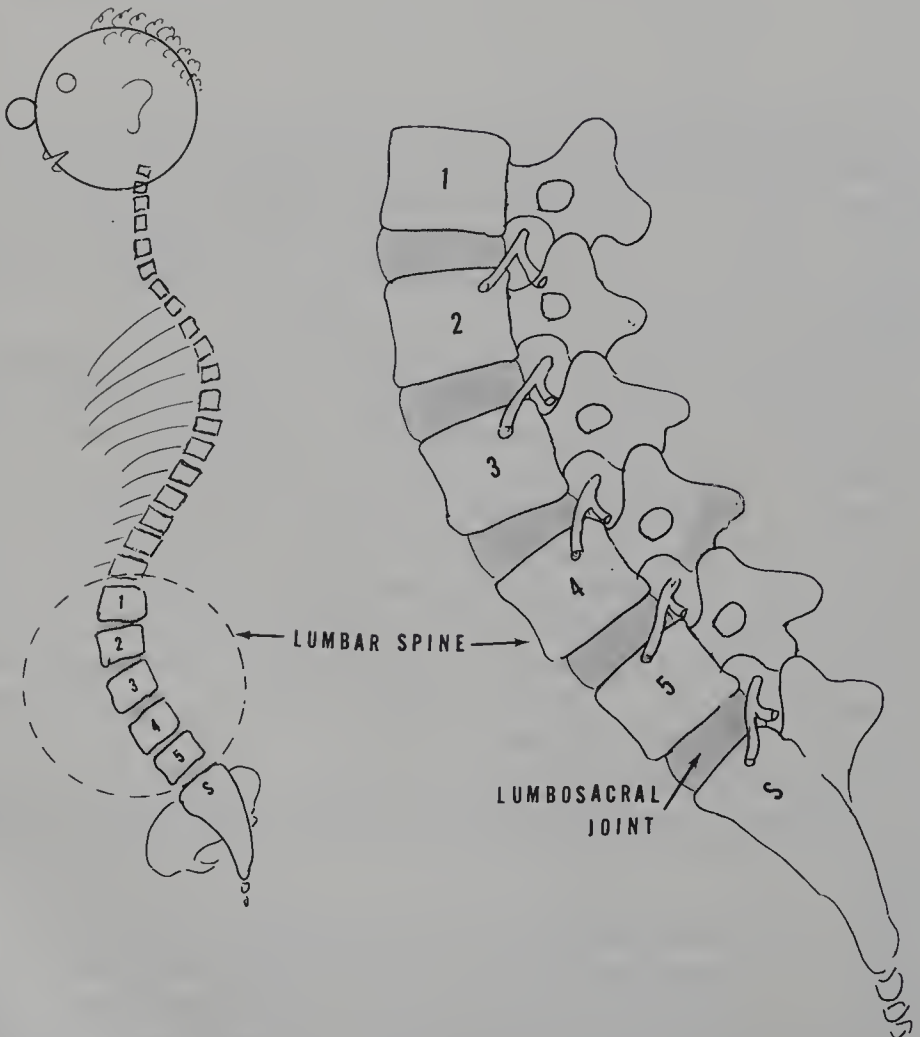
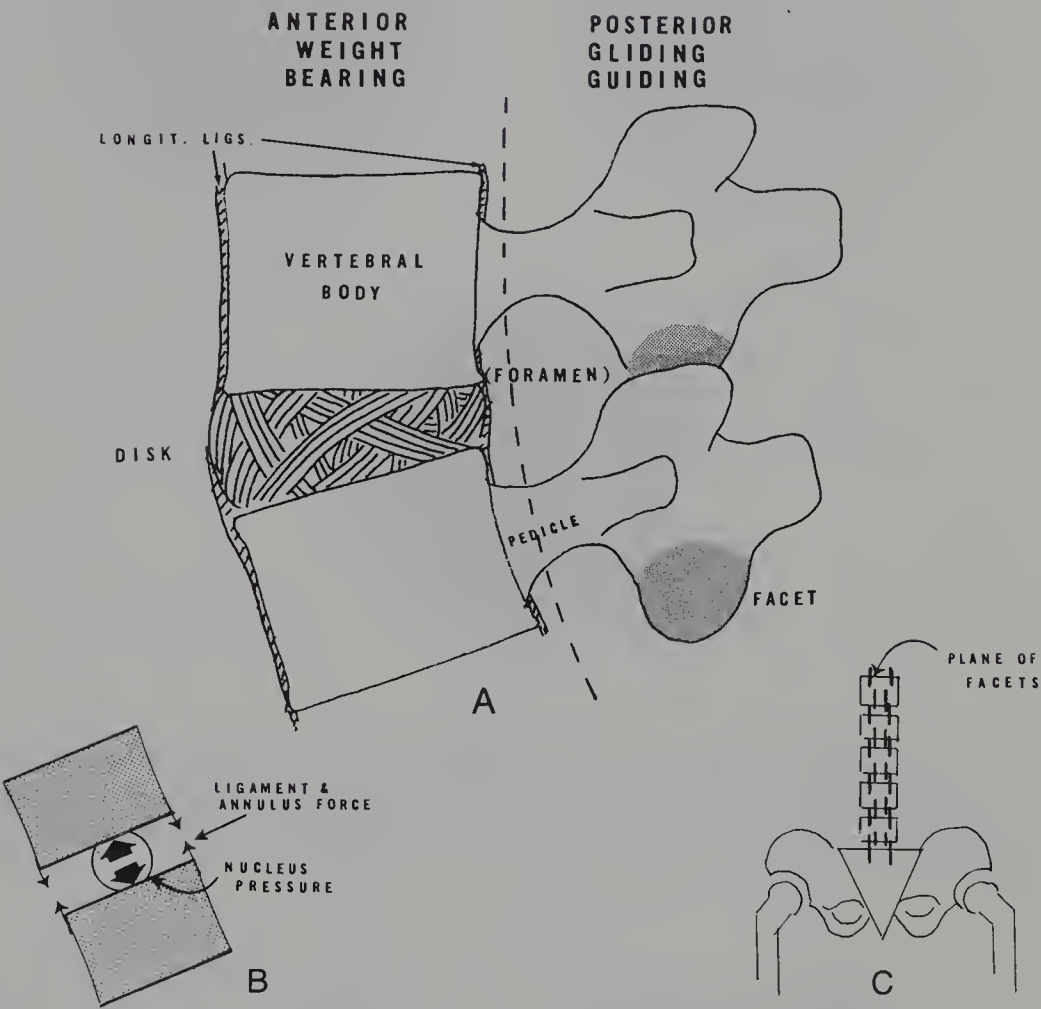


Figure 1-2. Lumbosacral spine. The lumbosacral spine usually consists of five functional units. Occasionally some functional units are fused into one and rarely there are six functional units. (Modified from Cailliet, R: Understand Your Backache. FA Davis, Philadelphia, 1984, p 5.)



**Figure 1-3.** The intervertebral disk. The side view of a functional unit showing the criss-crossing of annular fiber in the disk in the anterior weight bearing position of the unit (A). A schematic version of the nucleus, showing the force within the nucleus that separates the vertebrae (arrows). The annular and ligament forces (small arrows) contain the nuclear force (B). A sagittal view of the facets illustrated in the posterior gliding guiding portion of the functional unit (C).

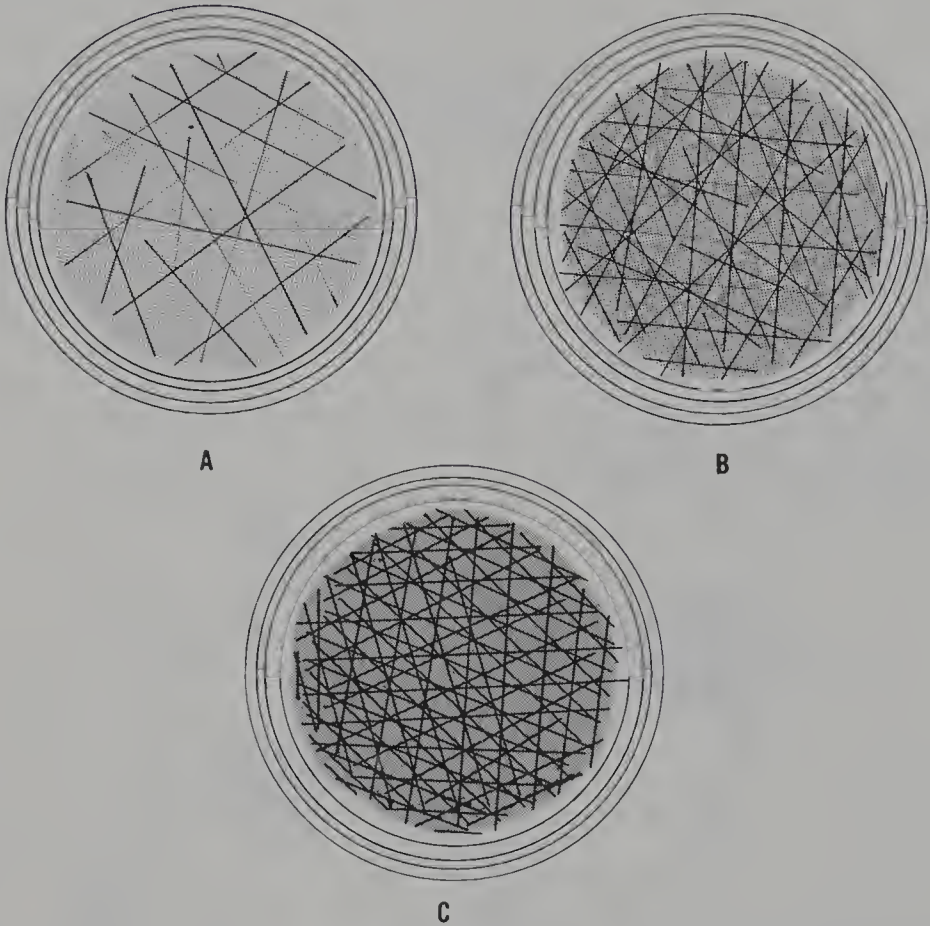


Figure 1-4. Collagen fibers of the nucleus pulposus. The nucleus is a mucopolysaccharide gel containing small irregularly directed annular fibers (A). As the nucleus ages (B) the fibers increase in size and number until they are the total content of the nucleus (C).

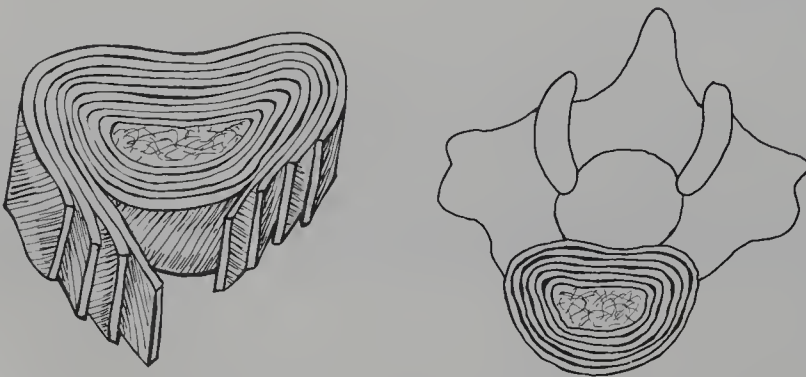


Figure 1-5. Annulus fibrosus. The annulus is a series of concentric annular fibers that run in opposite directions in an oblique manner in each layer. The fibers originate from one vertebral endplate into the opposite endplate. The annulus contains a pulpy nucleus (nucleus pulposus).



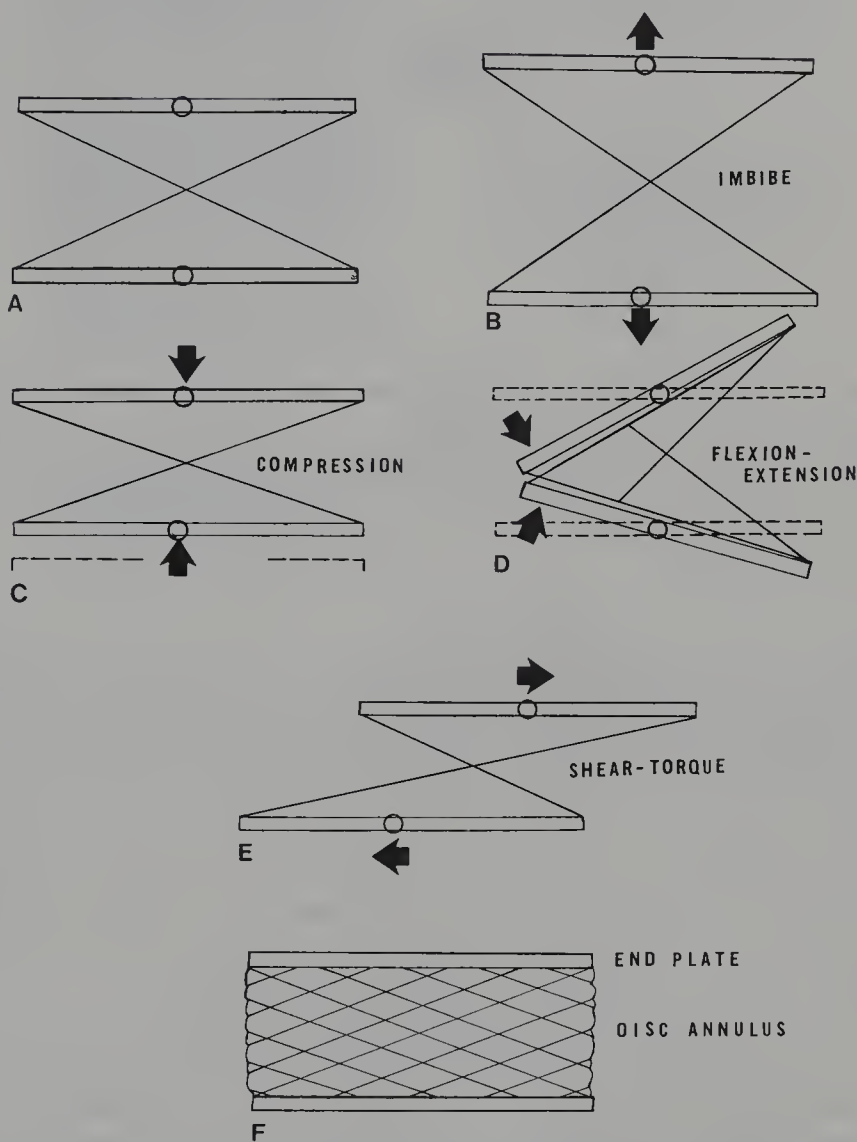


Figure 1-6. Annular fiber angulation under various forces. The angulation of the annular fibers with the intervertebral disk under no external pressure (A). The angle position is approximately 30 degrees. As the disk expands in imbibition the angle becomes less acute (B). Under external compression the angle becomes more acute (C). During flexion and extension of the functional unit, the fibers angle according to their anterior or posterior position (D). Panel E depicts the angulation and the length of annular fibers during shear or torque force. Panel F shows the attachments of the angled fibers with the endplates of adjacent vertebrae.

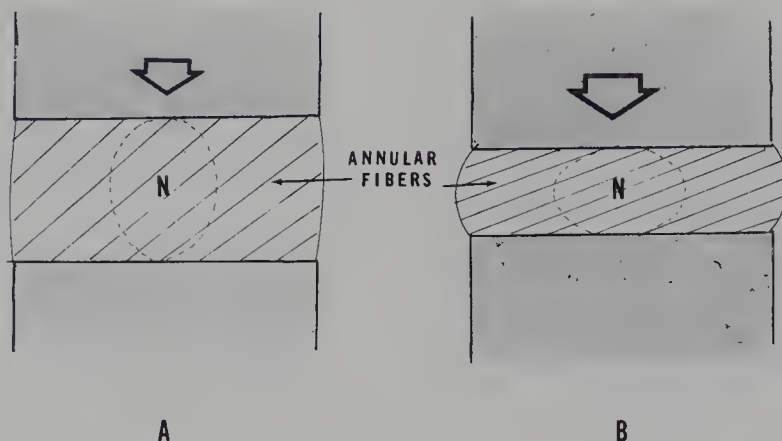


Figure 1-7. Change in shape of nucleus and angulation of annular fibers from compression. With a limited external pressure (*arrow*), the nucleus (N) retains its physiologic form and annular fibers angulate at approximately 30 degrees (A). With greater external force (*arrow*) the nucleus deforms and the fibers assume a more acute angulation and length. The disk physiologically bulges circumferentially (B).

significance as the fibers respond to the forces imposed on the disk. They elongate further from shear torque (rotation) than from mere compression, flexion-extension, or the combination. Compression does elongate the fibers, as does increase in the nuclear pressure (Fig. 1-7). Shear also increases elongation of the fibers due to their angulation. Maximum elongation occurs from compression, rotation, and shear. In flexion of the functional unit, the posterior fibers of the annulus also undergo greater elongation. These combined stresses are responsible for failure (tear or disruption) of the annular fibers.

The angulation of the annular fiber also varies, depending on its relationship to the centrum (nucleus). The angle becomes more acute as the center is approached (Fig. 1-8). This has clinical significance as the elongation of the fiber differs in its relationship to the nucleus and has a differential failure rate of the outer (longer) fibers to the inner (shorter) fibers.

The structure of the collagen fiber must be understood to determine its elongation, its retraction, and its length at failure. The fiber is a coiled chain of amino acids (Figs. 1-9 and 1-10) that uncoils when elongated and recoils when relaxed. Excessive elongation disrupts the bondings and denies return to its initial state.

The endplates to which the annular fibers attach are remnants of the growth plate, which in the young is fibrocartilage. This cartilaginous endplate gradually undergoes ossification primarily in a circumferential manner surrounding the endplate. This forms a ring around the end of



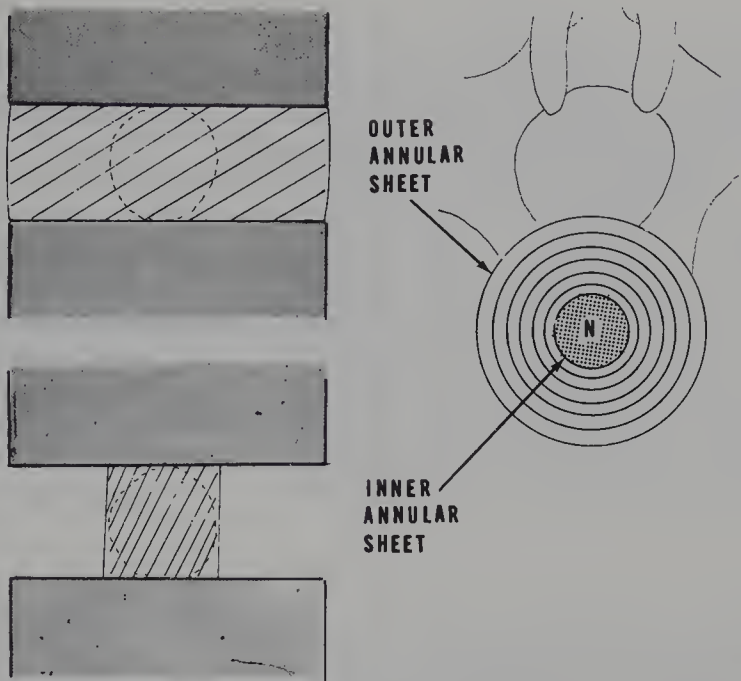


Figure 1-8. Variation of fiber angle. The right figure depicts the outer and the inner annular sheets that are portrayed in the left figures. The upper left shows the angulation of the fibers in the outer sheets, whereas the inner fibers (bottom left figure) forming the outer border of the nucleus are shorter and at a smaller angle. This is significant in stress reaction to shear and torque.

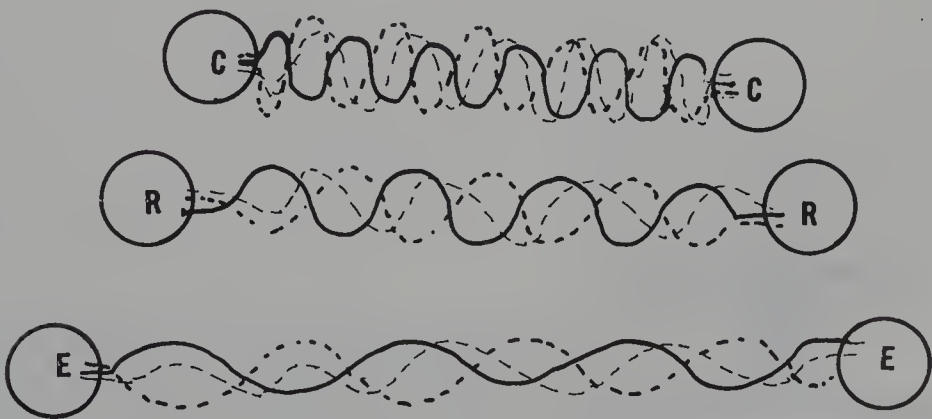


Figure 1-9. Collagen fiber (schematic). A collagen fiber (R) is a coiled trihelix chain of connected amino acid radicals (see Fig. 1-10). As the fiber elongates with moderate tension (E) the chain uncoils and elongates. It has a physiologic length that can elongate and uncoil before disruption. The fiber at rest fully coiled (C).

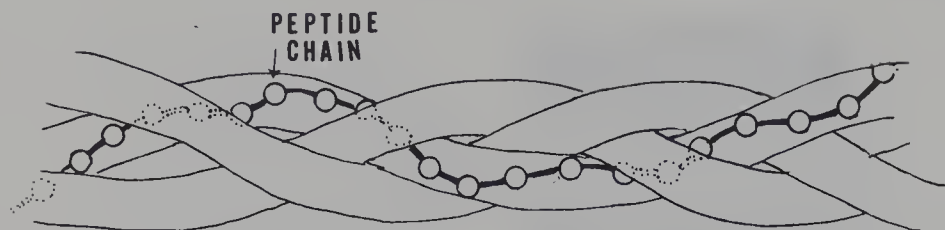


Figure 1-10. Tropocollagen trihelix fiber (schematic). This type I collagen molecule is composed of peptide chains that include two  $\alpha_1$  and one  $\alpha_2$  peptide chains in which every third molecule is a glycine amino acid. The three intertwining peptide chains form a trihelix collagen fiber. (Modified from Alberts, B, Bray, D, Lewis, J, et al: *Molecular Biology of the Cell*. Garland, New York, 1983, p 694.)

the vertebra with only the central portion of the endplate remaining (Fig. 1-11). Because of the ossification there is a diminution of nutritional effusion through the endplate to the intervertebral disk. The central collagen fibers of the annulus insert into the vertebral endplates, whereas the peripheral fibers insert into the apophysis on the vertebrae. These variable insertional sites also explain the tearing of the outer annulus of the disk following a shear injury. The outer fibers are principally type I, whereas the central fibers are type II.<sup>4</sup> The two types have biological differences that are yet to be clarified as to injury and repair.

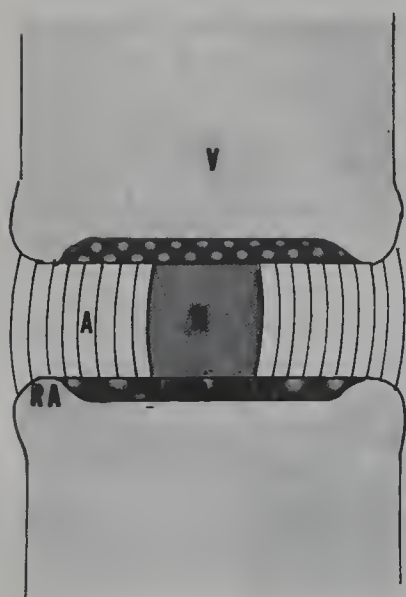
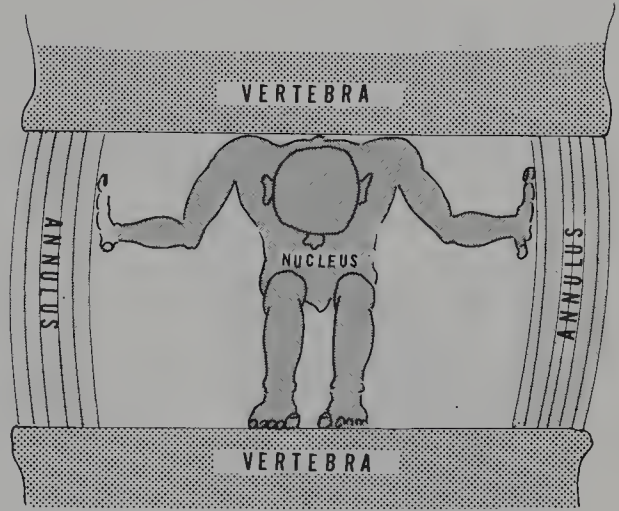


Figure 1-11. Structure of the vertebral endplate. The vertebral bodies (V) have their endplates as remnants of the growth plates (cartilage in the young). The ring apophysis (RA) is calcified. The nucleus (N) is centrally located and the annular fibers (A) attach to the endplates with the outer fibers attaching to the ring and the inner fibers attaching to the endplate.

Figure 1-12. Function of the nucleus pulposus (schematic). The nucleus pulposus has an intrinsic pressure that separates the vertebral endplates and keeps the annular fibers taut. (From Cailliet, R: *Understand Your Backache*. FA Davis, Philadelphia, 1984, p 17, with permission.)



Functionally the intervertebral disk separates the two adjacent vertebrae due to its intrinsic internal pressure (Fig. 1-12). This internal pressure not only separates the vertebral endplates but causes increase in tension of the annular fibers and ligaments of the functional unit. Increased gravity pressure increases the intradiskal pressure as does increased extravertebral muscle contraction. This hydrodynamic tension occurs within the disk but not exclusively within the nucleus. Removal of the nucleus does not impair compression loading but does allow increased “creep” of adjacent vertebrae and permits earlier failure from excessive external forces.<sup>5</sup>

The weight-bearing factors of the functional units have undergone intensive study, with the anterior portion of the units being considered the total weight-bearing position.<sup>6-8</sup> This concept assumes total weight bearing being rendered by the intrinsic disk structure and the longitudinal ligaments and no weight bearing from the posterior structures. The posterior structures, however, are now recognized as participating in weight bearing.<sup>9-13</sup> The effects of the posterior ligaments are still controversial even though they have been included in the anterior aspect of the functional unit.<sup>14</sup>

The physiological changes in the disk height on weight bearing remains unproved. Axial weight bearing in vivo at the L4-5 level as considered to be 1 mm in both directions,<sup>15</sup> with the rotational range being 8.4°.<sup>16</sup> Farfan et al.<sup>13</sup> reported failure of a normal functional unit to occur in the range of 22.6°.

Besides gravity on the superincumbent spine, the force exerted on the functional unit of the spinal muscles is difficult to calibrate. In vitro studies obviously do not include these forces, and in vivo forces are difficult to ascertain.

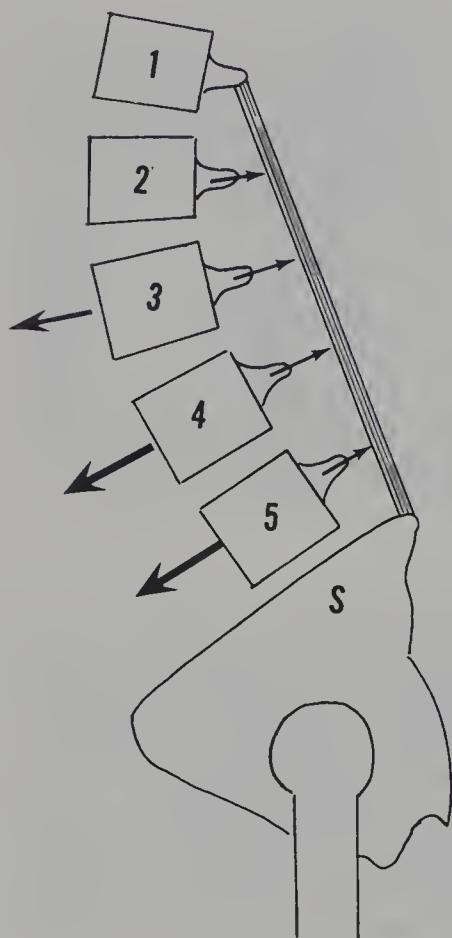


Figure 1-13. Shear stress on the lumbosacral spine functional units. The physiologic lordosis causes the vertebrae to assume an inclined plane orientation that causes shear translation (*large arrows*). The individual ligaments (*small arrows*) attaching to the posterior spinous ligaments minimize the shear force.

Recent studies have revealed that failure from shear displacement occurs differently in varying degrees of flexion (Figs. 1-13 and 1-14).<sup>17,18</sup> Besides the stiffness incurred by the annular fibers, there is evidence of stiffness imposed by the posterior elements both in compression and in torque.<sup>17</sup> The former is constant, whereas the latter increases on facetectomy. "The facets are involved in joint stiffness as are the annular fibers and are increasingly so with flexion shear forces."<sup>18</sup> The supraspinous and interspinous ligaments also have a significant effect on tensile stiffness.

The nutrition of the disk, annulus, and nucleus, is by imbibition (Fig. 1-15) as the blood vessels approach but do not penetrate the endplate and enter the disk. Intermittent compression and relaxation insure imbibition.

Before leaving the intervertebral disk completely, we must emphasize the following. Rotation limitation is imposed by only half of the annular fibers because, due to alignment, each layer (sheet) becomes elongated while the other becomes relaxed. Those that become elongated are those oriented in the direction of rotation. The limitation

Figure 1-14. Shear stress on facets. The facets or zygapophysial joints (*arrows*), restrict forward translation of the superior vertebra on the inferior vertebrae. A greater lumbosacral angle increases the shear stress. The posterior ligaments are slack in this degree of lordosis.

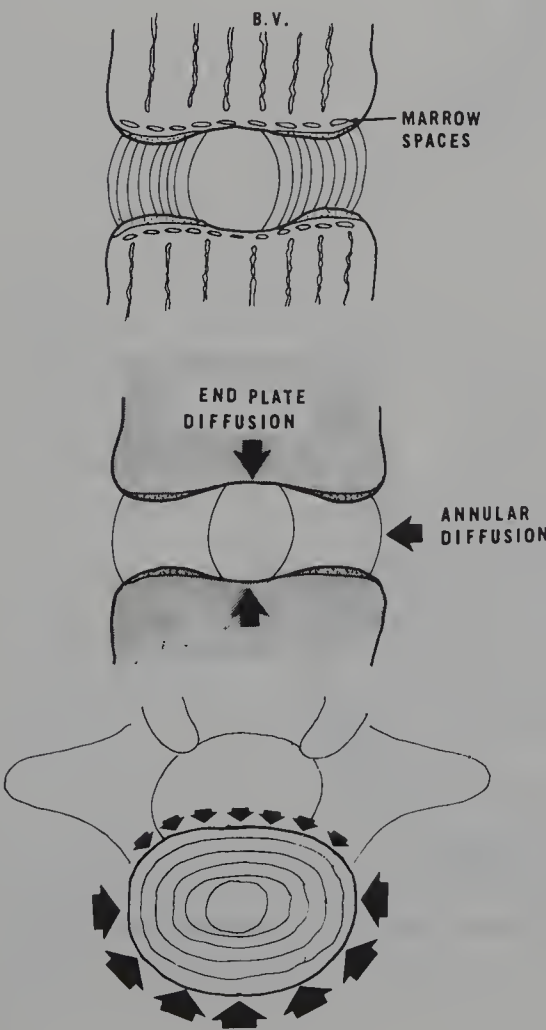
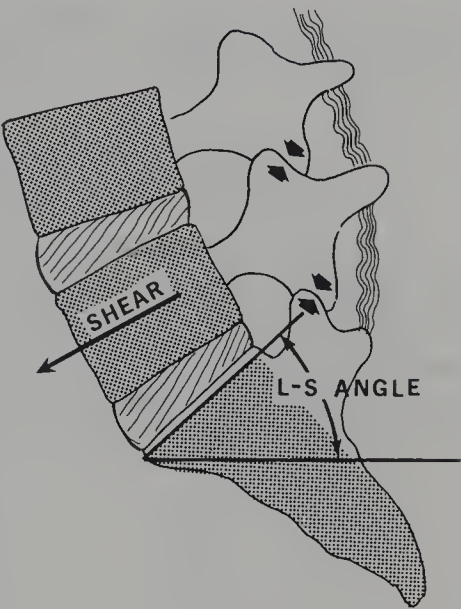


Figure 1-15. Disk nutrition through diffusion. Diffusion of solutes occurs through the central portion of the endplates and through the annulus. Marrow spaces exist between circulation and hyaline cartilage and are more numerous in the annulus than in the nucleus. Glucose and oxygen enter through the endplates. Sulfate, to form glucosaminoglycans, enters through the annulus. There is less diffusion into the posterior annulus. (B.V. = blood vessels.)



permitted by annular rotation has been estimated to be  $3^{\circ}$ , because collagen fibers allow only a 4% elongation before failure.<sup>4</sup>

## POSTERIOR SPINAL ELEMENTS

The anterior aspect of functional units has so far been emphasized but posterior to the vertebral bodies and their disks are the posterior bony elements that form the spinal canal (Fig. 1-16). Within the laminae the zygapophysial joints (facets) are contained. These are paired synovial joints articulating between the inferior aspect of the superior vertebra and the superior aspect of the inferior vertebra (Fig. 1-17).

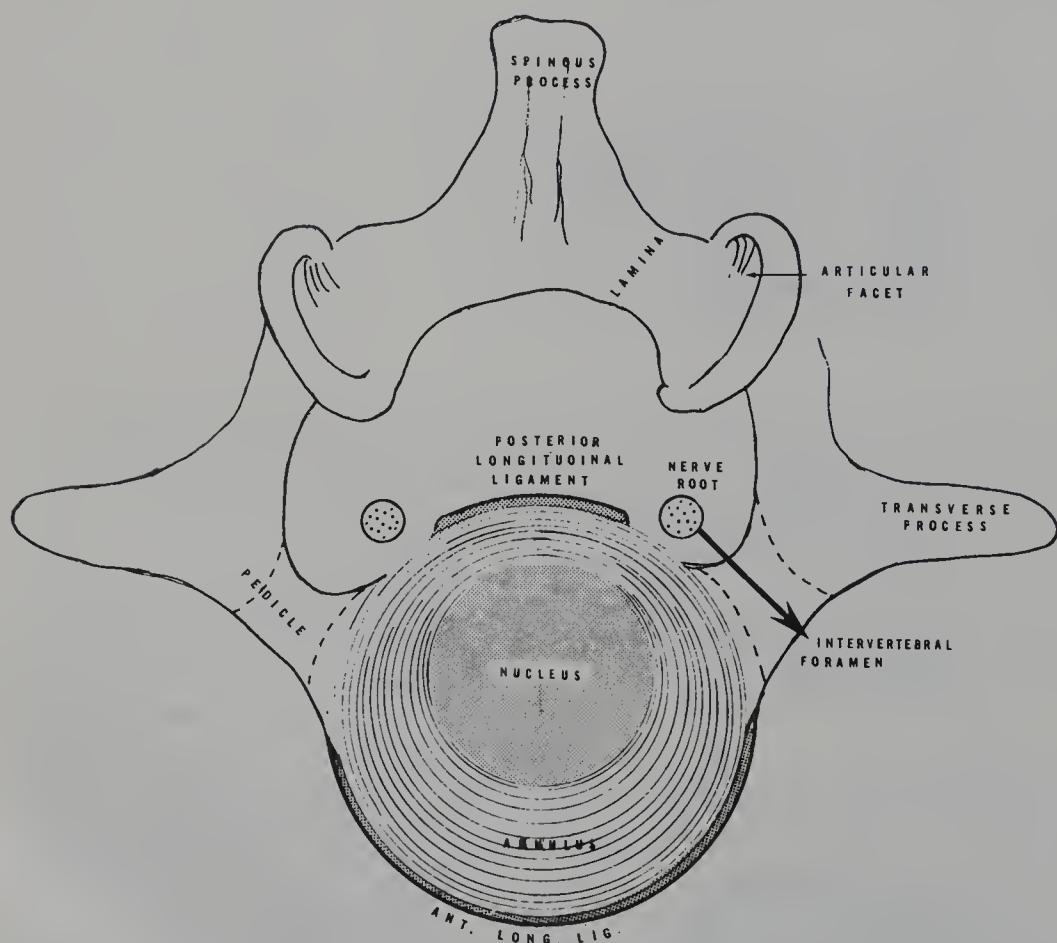


Figure 1-16. Functional unit viewed from above. Components of the functional unit as viewed from above.

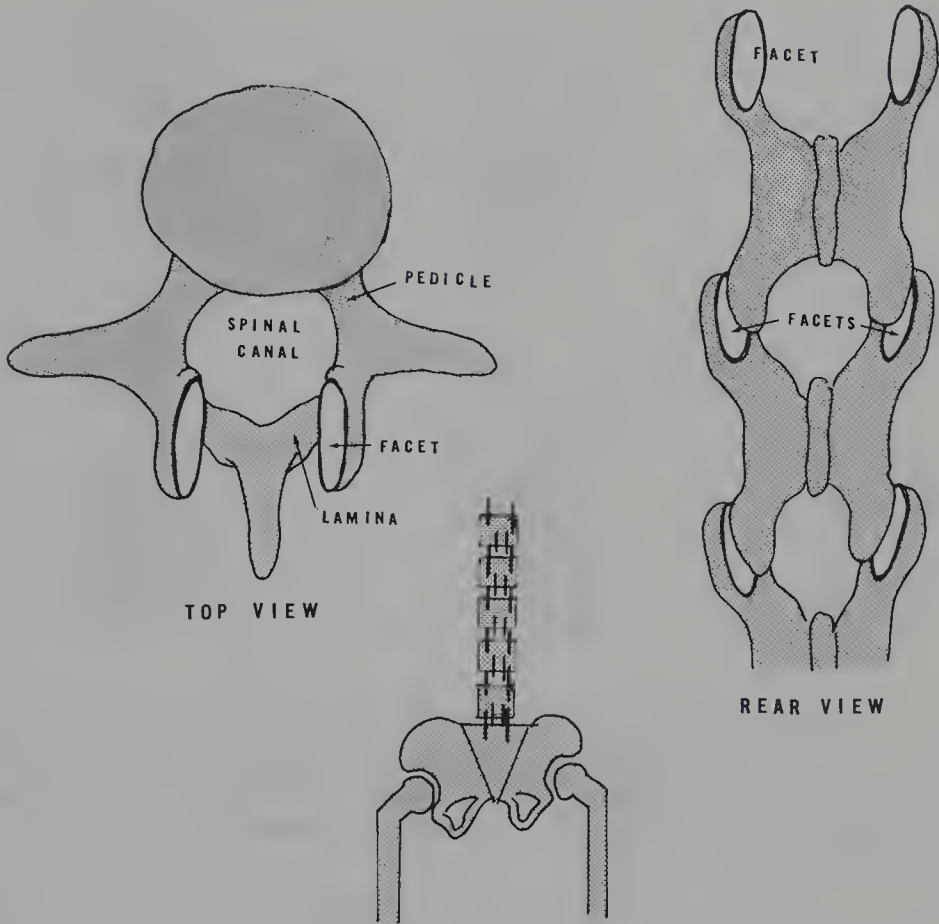


Figure 1-17. Facet alignment. Top view and rear view of the lumbar spine depicts the vertical alignment of the facets that permit flexion and extension and limits lateral and rotatory movement. The lower middle figure shows a schematic view of the facets.

By their sagittal plane the facets allow, in fact mandate, flexion-extension motion and restrict lateral (Fig. 1-18) flexion and rotation. The capsule that encloses the facet joints is redundant, allowing full flexion and extension. Ventrally the capsule is partially formed by the ligamentum flavum, which allegedly prevents entrapment of the synovium during these motions.

At their facet attachments the synovium is thickened and replicated on itself. It contains small fat pads that project into the joint space by several millimeters, forming a fibroadipose meniscuslike structure. The clinical significance that has been attributed to these “menisci” is that they may cause a facet impingement when they, after full trunk flexion,

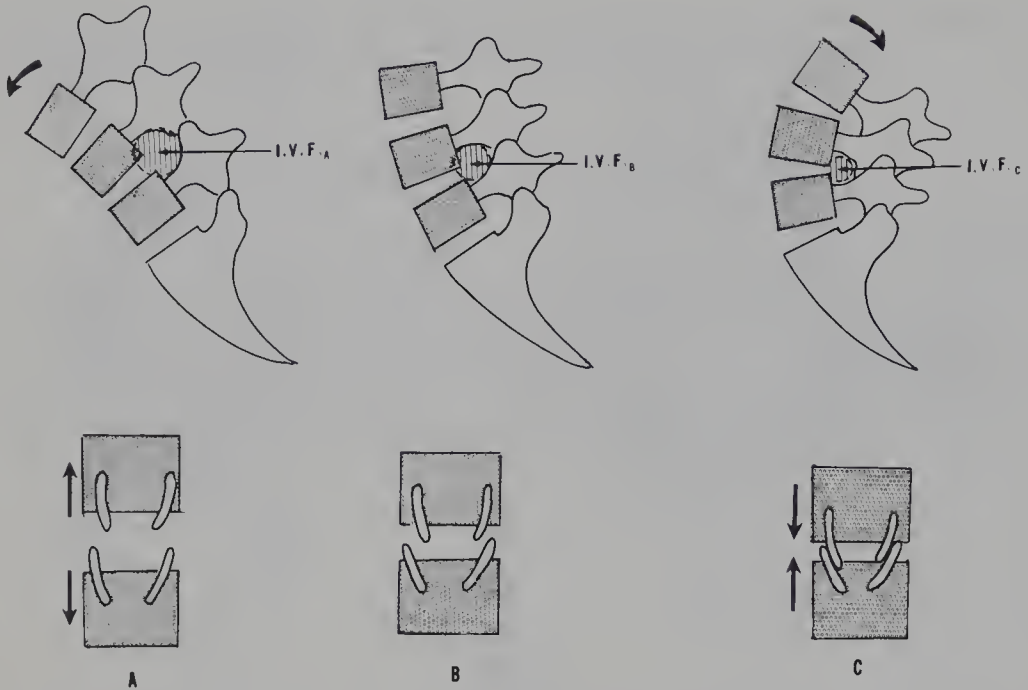


Figure 1-18. Facet movement in flexion and extension. The central figure (B) shows the neutral lumbosacral spine in physiologic lordosis with the facets slightly separated and the foramina (IVF) open. In flexion the facets separate and the foramina open further (A). In extension the facets approximate and the foramina narrow (C).

reenter the joint space on reextension. They also may be the nidus for adhesions forming between joint surfaces. The menisci are well innervated with proprioceptive nerve endings and nociceptor fibers thus they function for position sense and as a site of pain.

The facets are curved and obliquely oriented as well as being sagittal. They prevent or minimize translation (shear) motion of adjacent vertebrae. They prevent "listhesis" and, when defective, allow the condition of spondylolisthesis.

By limiting rotation, they minimize the rotational forces on the disk annular fibers, which have been indicted as the cause of annular fiber tearing (disk herniation or tear). They do, however, play a role in annular injury when proper neuromuscular mechanics are violated.

Flexion and reextension are always accompanied by some degree of rotation.<sup>19</sup> As a person bends forward and undergoes some degree of lateral flexion, there is also some degree of rotation (Fig. 1-19). The spine rotates away from the axis of rotation toward the convex aspect of the spine. The facets on the convex side open, and those on the concave side approximate (Fig. 1-20).



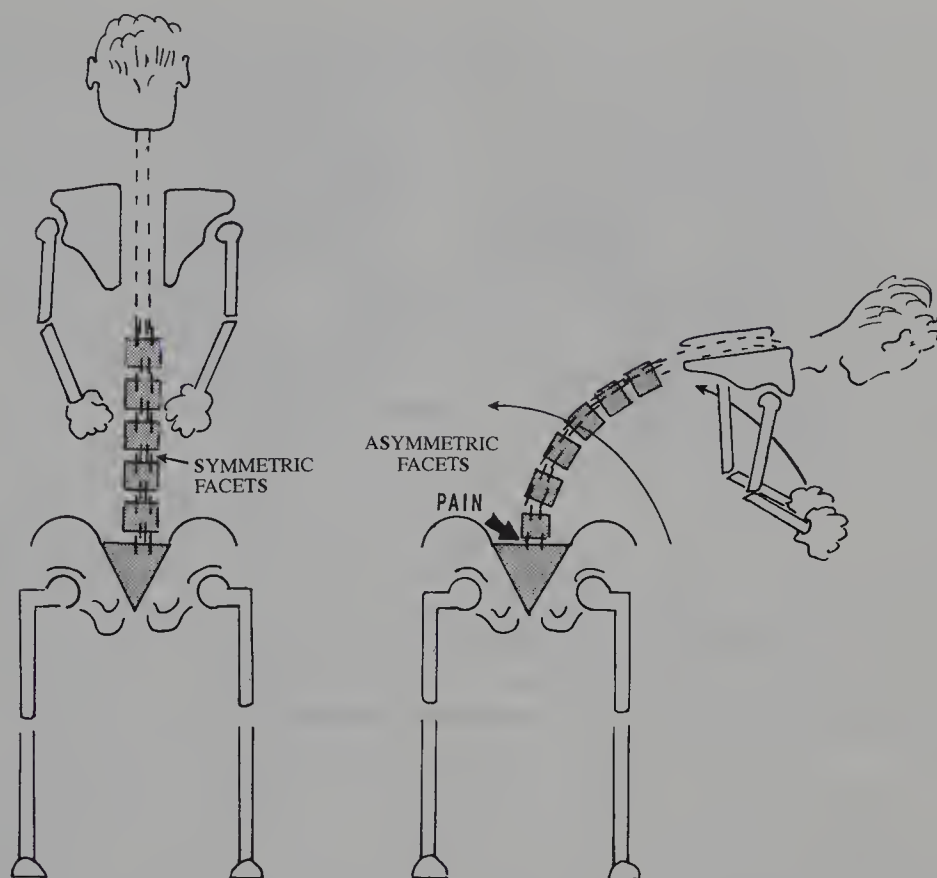


Figure 1-19. Facets during flexion and reextension. The figure on the left shows the facet alignment in a person standing erect. The figure on the right shows flexion, lateral flexion, and rotation in forward flexion to one side, which can cause impingement of the facets causing pain on reextension if the proper path of extension and derotation is not followed.

If a person violates this normal activity and does not adequately derotate, the facets on the concave side engage as they reextend and become the axis of rotation (derotation in this instance) (Fig. 1-21).

## LIGAMENTS IN SPINAL FUNCTION

The ligaments of the functional units have been found to play an increasing role in spinal function. The ligamentum flavum is 80% elastin and 20% collagen, which allows considerable elasticity (elongation) before failure. The supraspinous ligament connects the dorsal tips of the spinous processes of the individual vertebrae. It is irregular in its structure and presence so that its function is questionable. The interspinous

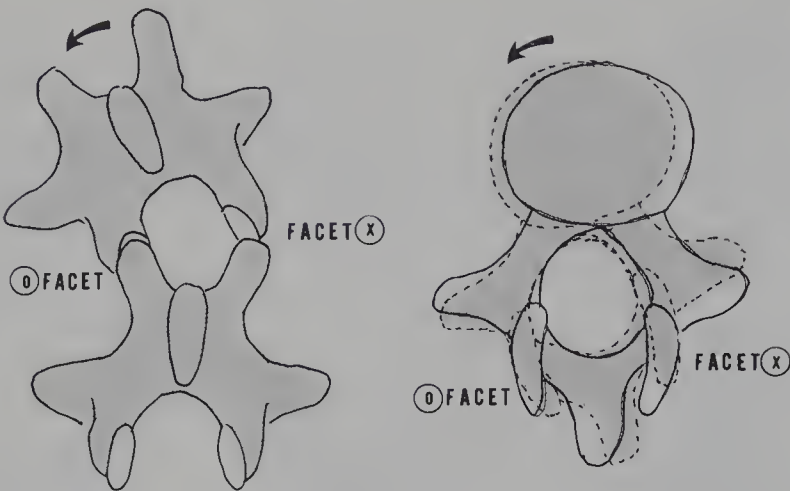


Figure 1-20. Lateral flexion rotational torque. When a person bends sideways the facets on the concave side (O) approximate and those on the convex side (X) open. The superior vertebra rotates (*curved arrow*) around facets (O) and becomes the axis of rotation. The figure to the right shows the final stage of physiologic rotation and lateral shear. The shaded vertebra is the superior vertebra, and the clear picture is the inferior vertebra.

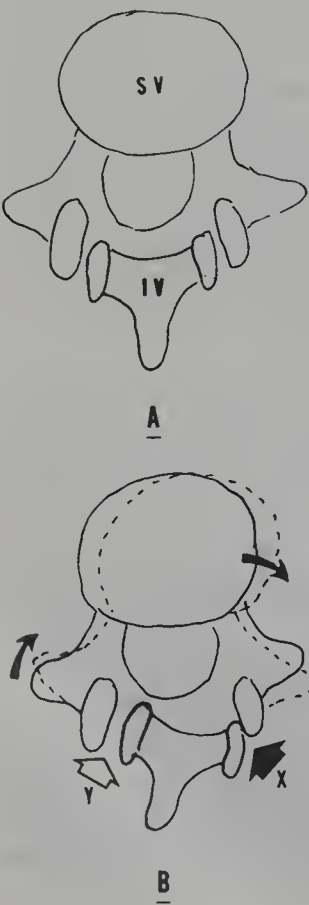


Figure 1-21. Axial rotation of a lumbar functional unit. The normal relation of adjacent vertebrae and their facets (A). (SV = superior vertebra; IV = inferior vertebra.) The impingement on abnormal derotation in reextension with X (*dark arrow*), showing impingement of the concave facets that become the axis of rotation causing excessive lateral shear (*curved arrows*) of adjacent (shaded) vertebra (B). The convex facets (Y) gap excessively. The inferior vertebra is the shaded drawing. The intervertebral disk now undergoes lateral and rotational shear.

connects the adjacent spinous processes and is divided into three portions: ventral, middle, and dorsal. It has more collagen hence less extensibility. The interspinous ligament connects the adjacent transverse processes and from its structure is questionably a mechanical support. From their structures the function of ligaments in regards to support and limitation of the spine remains questionable.

The posterior longitudinal ligament covers the posterior aspect of the lumbar vertebrae and is essentially an outer annular sheet. It diminishes in its width as it descends toward the sacrum (Fig. 1-22).

The multifidus triangle is an arbitrary triangular area that contains numerous nociception sites: facets, facet innervation, transversus ligament, quadratus lumborum muscle, and the iliolumbar ligament (Fig. 1-23). The nociceptive tissues contained are innervated by the posterior branch of the lumbar dorsal ramus.

## MUSCLES IN SPINAL FUNCTION

The spinal stability system will be discussed, but the erector spinae muscles comprising a major subsystem need clarification.

The erector spinae muscles, which flex the trunk (especially the lumbosacral portion) have undergone serious studies, varying from the deceleration of the erector spinae muscles during active flexion to acceleration in reextension. The forces expended on the functional units is undergoing an increasing number of studies. As well as controlling flexion-extension, the erector spinae muscles exert compressive and shear forces on the functional unit.

The configuration, direction, and angulation of the erector spinae muscle fibrils in the erect posture have been well documented by Bogduk et al.<sup>17</sup> More recently, studies have been made of the flexing spine.<sup>18</sup> In the erect posture the angulation of the fibrils varies according to the degree of lordosis and the distance from their axis of rotation as well as to the specific unit on which they are acting. In flexion this alignment changes with every degree of angulation of each functional unit as the spine progressively flexes (Fig. 1-24).

In flexion there is a significant elongation of the erector spinae fibrils and changes in their orientation to the spine. As the multifidus and the iliocostal-longissimus crisscross during flexion, they vary in their alignments and essentially do not change in their torque or compressive forces.<sup>7</sup> The elongation of the fibril (15% to 59%) reduces their active tension but increases their passive tension, which explains this paradox. In the erect posture the fascicles of the multifidus are oriented in a dorsocaudal position but become ventrocaudal in flexion, whereas the fascicles of the longissimus merely align more to the longitudinal axis of the spine.

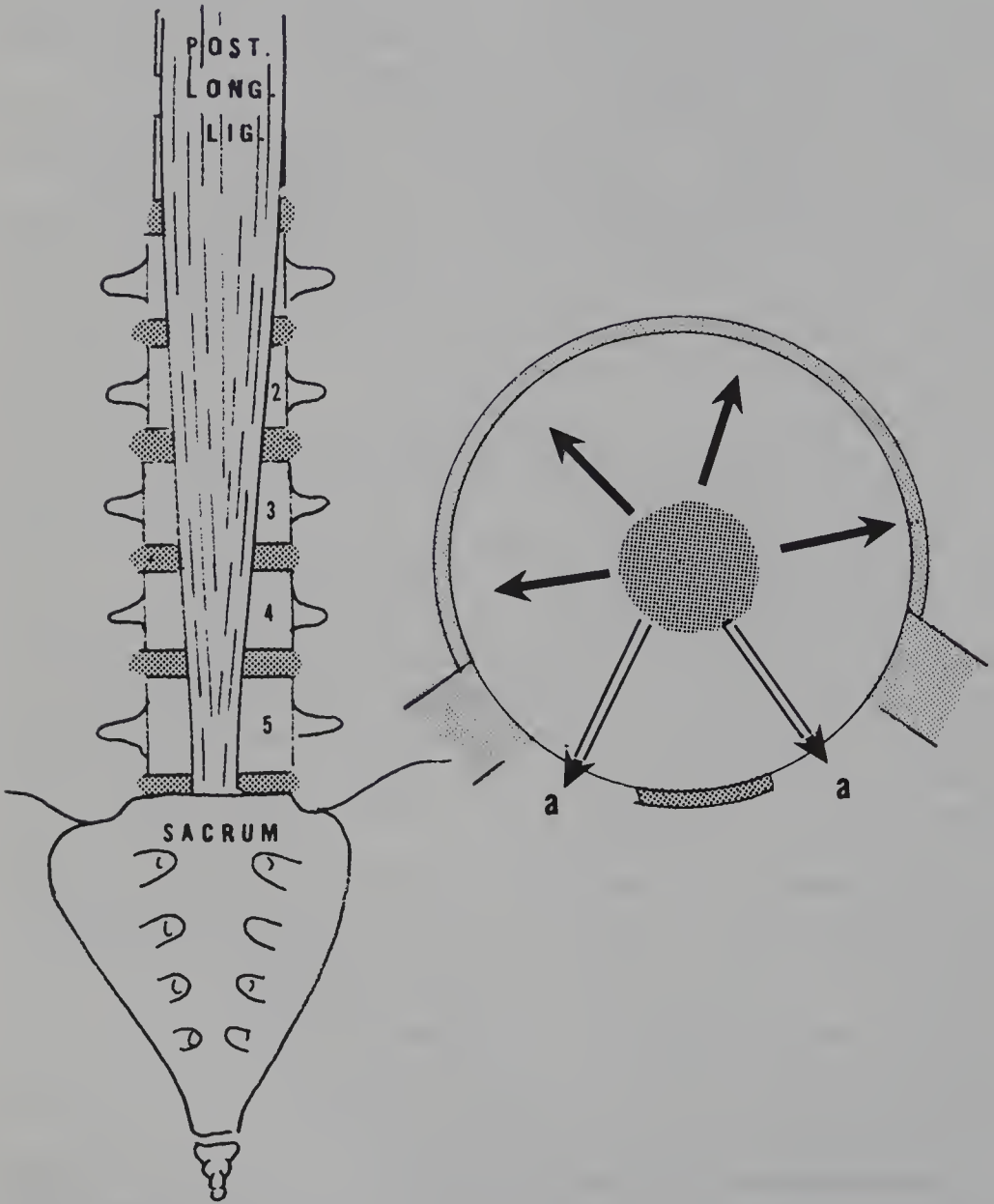
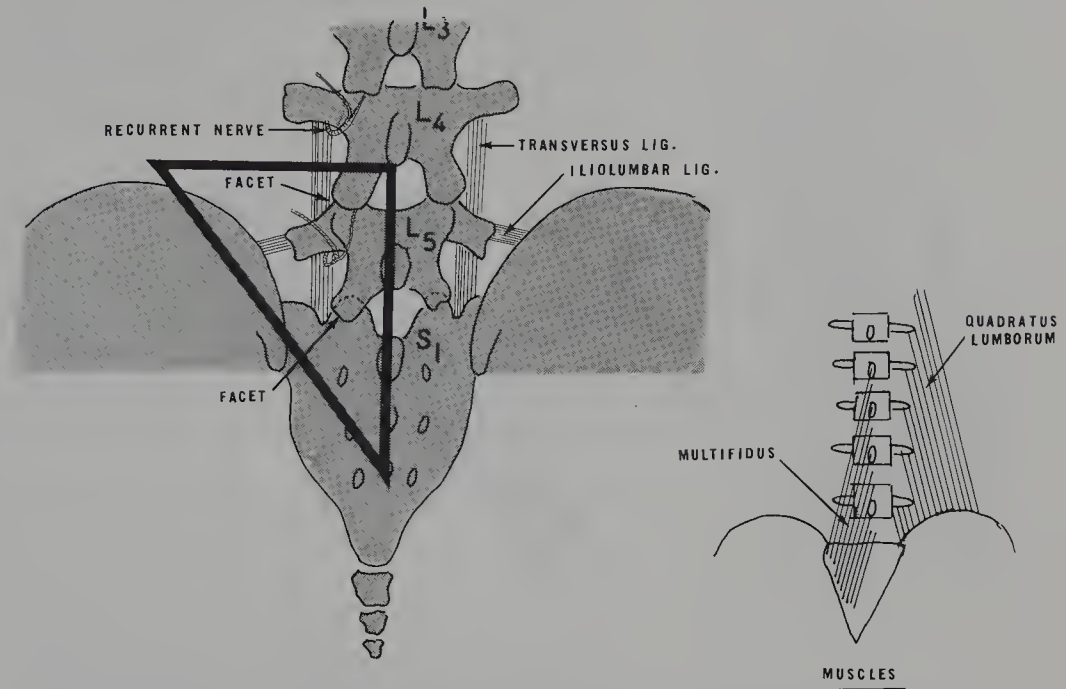


Figure 1-22. The posterior longitudinal ligament. The posterior longitudinal ligament, which attaches to the entire posterior aspects of adjacent vertebrae within the spinal canal, narrows as it descends and is only partial at the L5-S1 levels.



**Figure 1-23. Multifidus triangle.** The quadratus lumborum muscle is a large muscle lying lateral to the multifidus muscle. Its fibers attach to the transverse processes and the iliac crest. The multifidus triangle includes the transverse process of the fifth lumbar vertebra, the facets of the two lower lumbar functional units, the transverse ligament from the fifth lumbar transverse process to the iliac crest, and the recurrent nerve of the L4-L5 foramen. All of these tissues are nociceptive sites.

In summary the geometric changes of the erector fascicle orientation do not alter the strength (torque) nor the compressive forces of the muscle during flexion.<sup>7,18</sup>

## Intersegmental Muscles

The short intersegmental muscles (Fig. 1-25), interspinalis and intertransversalis, are too short to exert any significant force in extension or deceleration. Their force in rotation is unproved. They are usually well developed, so they must function in spinal movements.

## Quadratus Lumborum Muscle

The quadratus lumborum muscle is a sagittal muscle that originates and inserts from the twelfth rib, the iliac crest, and the transverse



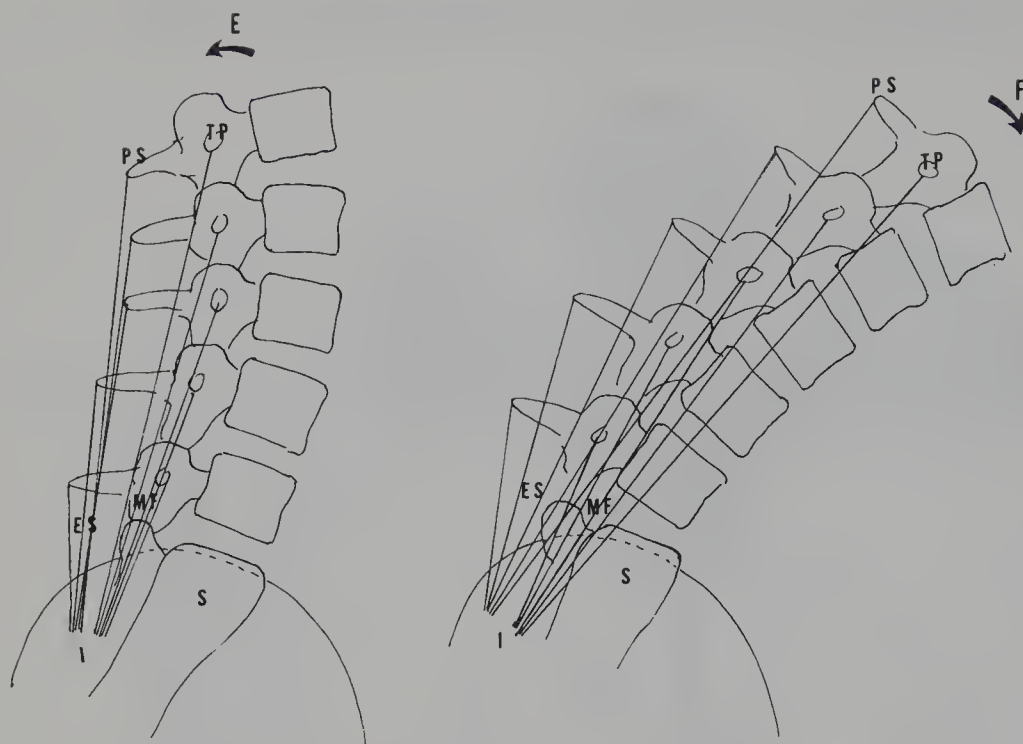


Figure 1-24. Extensor muscles in trunk flexion-reextension. In the erect posture (E) the erector spinae muscles (ES) and multifidus muscles (MF) generate specific force and have a specific length and angulation. The erector spinae muscles attach to the posterior spinous processes (PS) and the multifidus muscles attach to the transverse processes (TP). In the flexed trunk position (F) the erector spinae and the multifidus elongate and generate less force, but this is balanced by the elastic increase of the elongated muscle as they crisscross. The generated force is equal in the erect posture to the fully flexed. These forces are both eccentric and concentric.

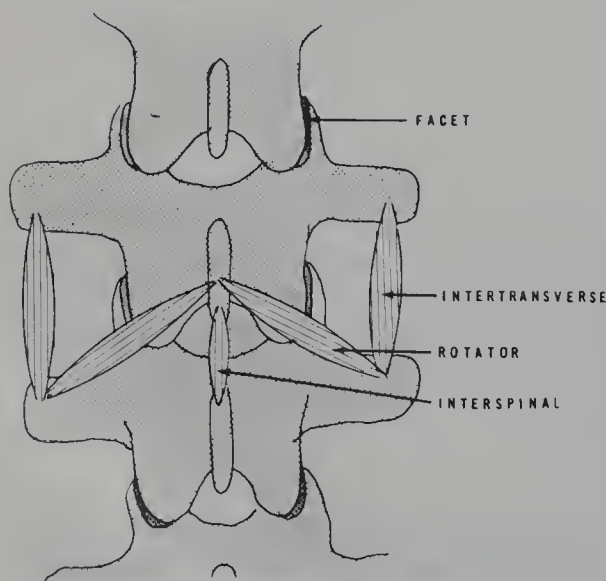


Figure 1-25. Deep intersegmental muscles. The deep intersegmental muscles are extensors, lateral flexors, and rotators by virtue of their attachments.



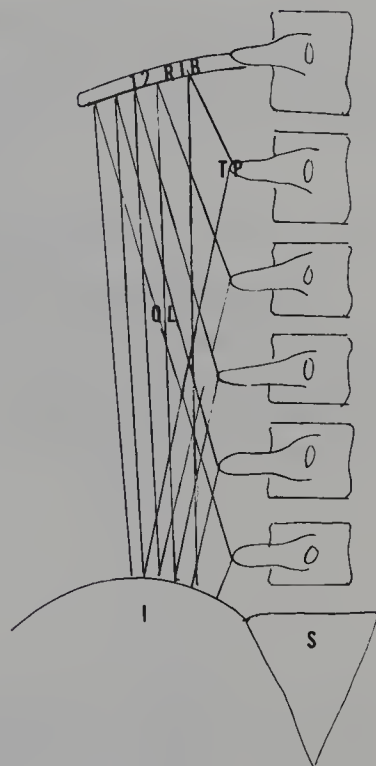


Figure 1-26. Quadratus lumborum muscle. The quadratus lumborum muscle (QL) attaches from the iliac crest (I) to the transverse processes (TP) of the lumbar spine and the 12th rib. S refers to the sacrum.

processes of the lumbar vertebrae (Fig. 1-26). This muscle is considered a lateral trunk flexor, and the upper longitudinal fibers are essentially respirator muscles.

## Multifidus

The multifidus muscles are termed in the plural because they are segmental in structure. They stem from the laminae of the lumbar vertebrae and the spinous processes attaching to the sacrum. They are short muscles that act in a sagittal plane: flexing and extending the spine.

## Erector Spinae Muscle

This is a large muscle lying lateral to the multifidus that extends into the thoracic region. It contains several divisions that, by their names, indicate their sites of attachment: longissimus thoracis and iliocostalis lumborum. These fiber groups attach to the transverse process of the vertebrae and the iliac crests. The thoracis fibers attach to the transverse processes of the thoracic vertebrae and the ribs.

By their attachments to the transverse planes of the vertebrae, they exert no posterior translation of the functional units but merely sagittal motion and rotation.

## STATIC SPINE

The nonmoving erect spine can be considered the *static spine*. In the erect stance it is termed *posture* and consists of four physiological curves: cervical, thoracic, lumbar, and sacral. The cervical and lumbar curves are termed *lordotic*, and the other two *kyphotic*. All conform to the center of gravity (Fig. 1-27).

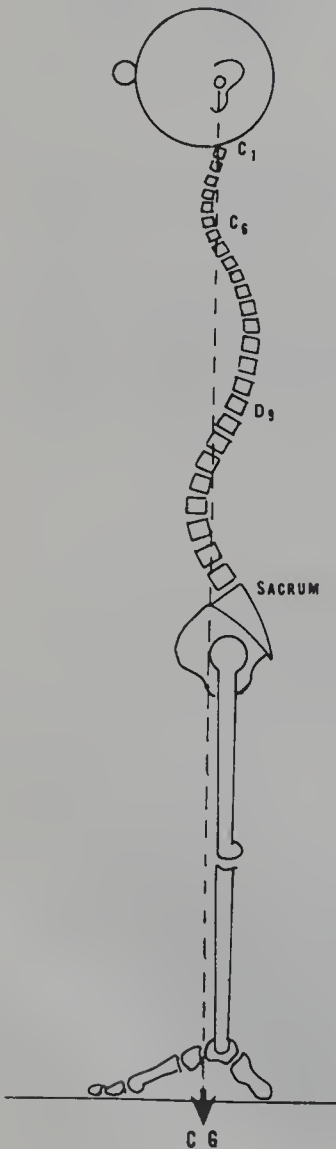
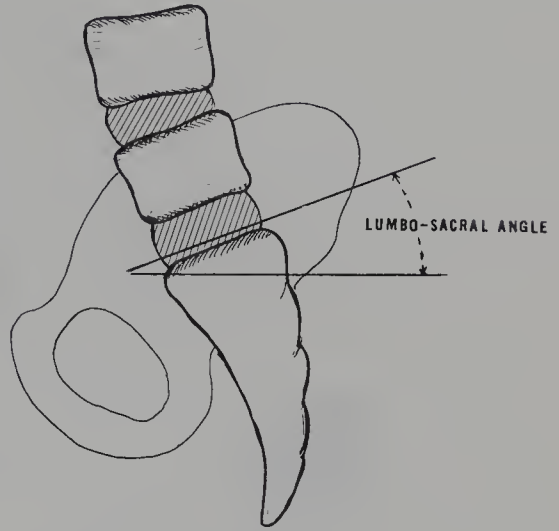


Figure 1-27. Physiologic curves of the erect spine. The physiologic curves of the erect spine transect the center of gravity (CG), which passes anterior to the hip articulation. The curves are the cervical (C), dorsal (D), and lumbar (L). The sacrum is the fourth (kyphotic) curve.

Figure 1-28. Physiologic lumbo-sacral angle. The lumbosacral angle is computed as the angle from the horizontal aspect of the sacrum to a line drawn parallel to the superior aspect of the sacrum. The angle should measure at approximately 30 degrees in healthy individuals.



The base of the spine is the sacrum, upon which the lowest lumbar vertebra resides. The angle of the sacrum determines the degree of the superincumbent lordosis. There is controversy about the physiological lumbosacral angle (Fig. 1-28). As the sacral angle changes, so does the lumbar lordosis (Fig. 1-29).

The pathological significance of the increased lordosis from an increased sacral angle will be discussed later, but suffice to say that its significance is now questioned. There is some shear stress of the lowest lumbar vertebra on their facets. This is physiological (see Fig. 1-13) and of questionable pathological significance.

The mechanical support of the erect spine has been accepted as being internal forces (intrinsic disk pressure) creating the stiffness with the external muscular elements being nonoperational. The external ligaments and facet capsule contribute to the stiffness.

In the erect static spine, the ligaments of the passive subsystem<sup>20</sup> do not provide stability in the neutral position. The collagen fibers of the ligaments remain coiled and under no tension as they are when uncoiled (see Figs. 1-9 and 1-10). It is only toward the end of their ranges of motion that ligaments develop reactive forces that resist spinal motion (Fig. 1-30). At these points in range of motion the tendons act as transducers (signal-producing devices) that initiate the motor aspect of the active subsystem via the neural subsystem. It is probable that this subsystem operates even during the minimal stresses occurring in the static spine.

The intervertebral disk (within the passive subsystem) passively acts to keep the vertebrae apart (Fig. 1-12), placing the annular fibers and the longitudinal ligaments under tension.

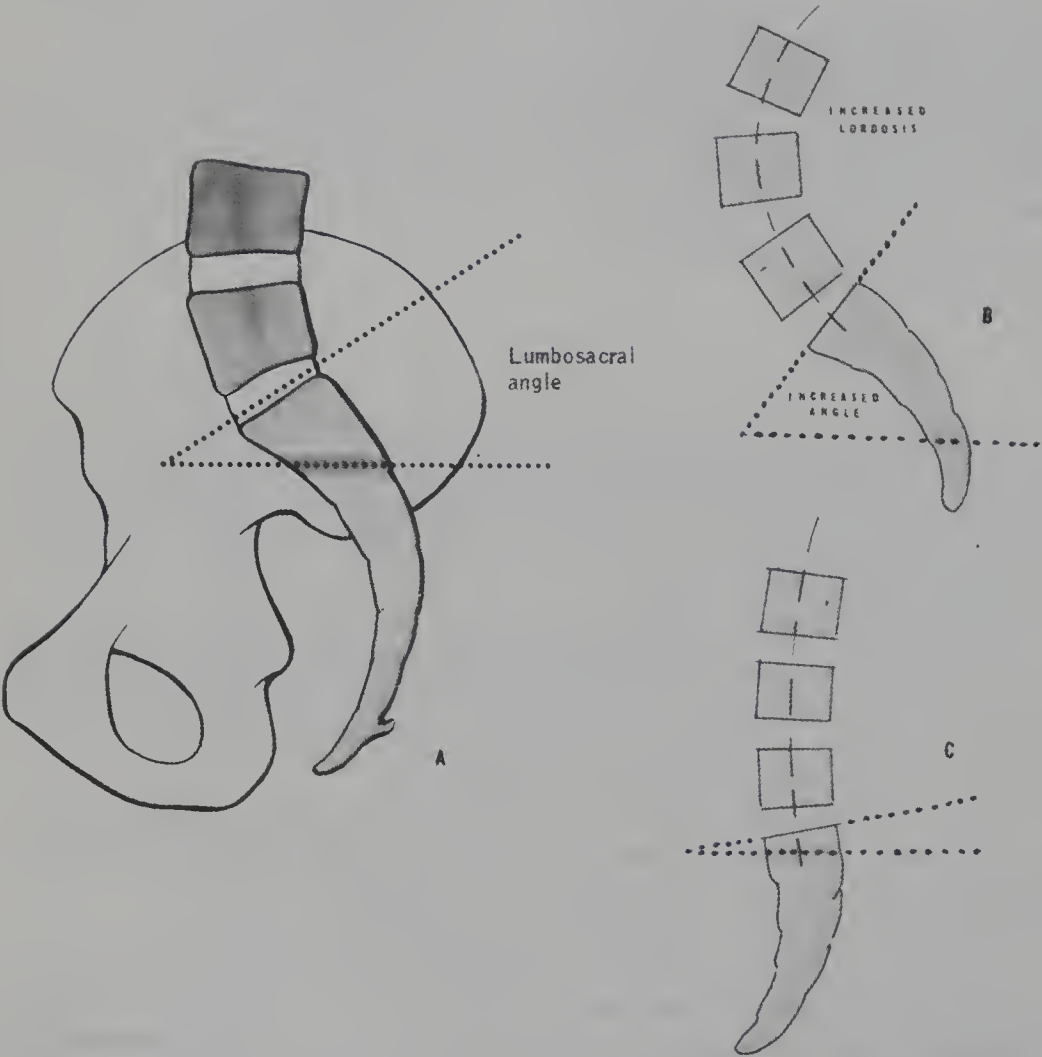


Figure 1 – 29. Variations of lumbosacral angle dependent on the sacral rotation. The physiologic lumbosacral angle is shown on the left (A). With an increased angle (B) the superincumbent lordosis is increased, whereas a decreased angle (C) decreases the lordosis.

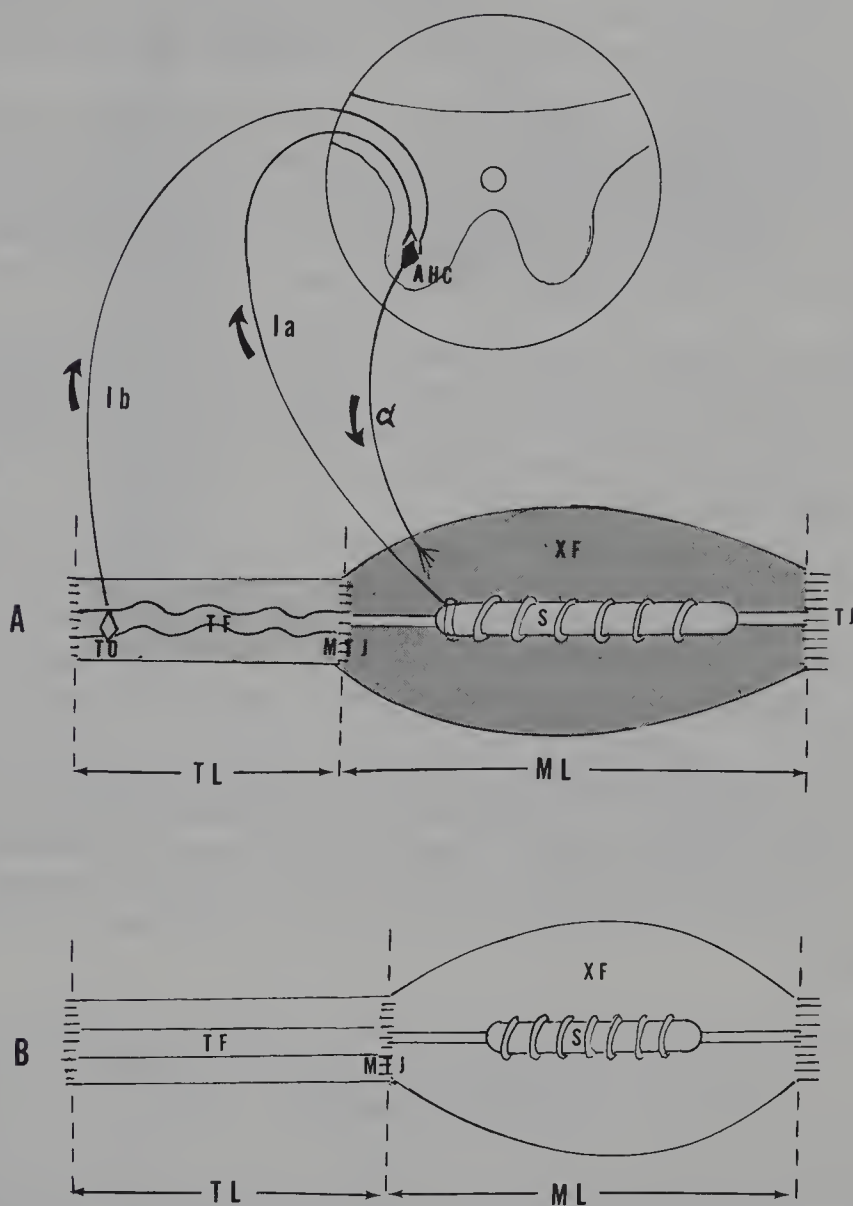


Figure 1-30. Musculo-tendinous mechanism. (A) The Spindle system (S) measures the length of the muscle (ML) and the tendon organs (Golgi) (TO) monitor the tension. Stretching the spindle system activates the Ia fiber whereas stretching the tendon organs activates the Ib fibers. These influence the anterior horn cell (AHC), which sends motor fiber activity via the alpha fibers to the extrafusal fibers (XF). With the muscle at resting level (A) the tendon fibers (TF) are slightly coiled. When the extrafusal fibers contract (B) the muscle shortens (ML), and the tendon elongates (TL) to the degree that the tendon fibers can elongate. The tendon fibrils (TF) uncoil. Excessive muscle contraction can tear the muscle-tendon juncture (MTJ).

The muscles in the active subsystem, under the control of the neural subsystem, remain in a state of tonus, which exerts sufficient tension within the intervertebral disk to maintain balance and thus stability.

The stable spine concept envisions a normal coordinated systems complex that adjusts instantly and appropriately to the external demands of function.<sup>20</sup> There are compensating forces unleashed when the external demands are excessive or abnormal. The resultant tissue impairment allegedly causes pain and dysfunction and hence symptomatology.

The load that can be supported by the passive subsystem before buckling has been termed the "critical load of the spinal column." In vitro studies show that the buckling load of the spine occurs at 20N (2 kg) at T1 and 90N (9 kg) at L5, which amounts to two or three times the body weight (140 to 210 kg).<sup>21</sup>

The tissue's response to the approach of the critical load is muscular response to ligamentous deformation rather than load stresses.<sup>22</sup> Deformation of ligaments thus provides a meaningful feedback mechanism to maintenance of spine stability. This feedback notifies appropriate muscular contraction via the spindle system and tendon organs (Fig. 1-31).<sup>23</sup>

The neural subsystem acts continuously and instantaneously, monitoring and adjusting and readjusting the subsystem as it reacts to forces acting on the vertebral column. The plasticity of the neural subsystem to adjust to normal<sup>24</sup> and abnormal forces and be modified by training<sup>25-27</sup> or restructured after injuries is of paramount importance in low back pathology.

## MUSCULAR SUBSYSTEM: ACTIVE AND PASSIVE

The muscular action plays a vital role in both passive and active subsystems. Biomechanical studies of forces exerted in various positions and activities have been computer-model studies (Fig. 1-32).

Early studies of back muscles were devoted to single force equivalents, whereas more recent studies show muscle to be multisegmental with numerous fascicles acting simultaneously in various directions and most acting through varying axes of rotation and varying distances from their line of action.<sup>27</sup>

The muscles of the back, which were originally relegated to forces acting in one direction between two points of attachment, now must be understood as multisegments acting on a given segment in numerous directions from various points of attachment thus on varying axes of



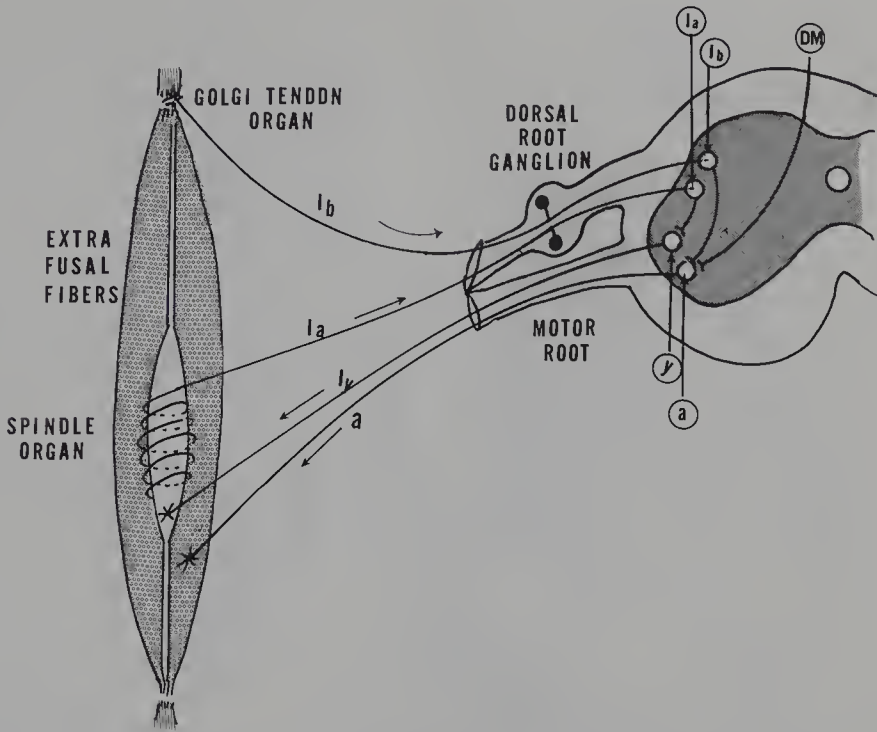


Figure 1-31. Proprioceptive autonomic control of neuromuscular function. The spindle system (intrafusal) of the muscle is innervated by Ia, Iy, and A fibers that originate in the gray matter of the cord. The Golgi tendon organ is also innervated by Ib fibers that ascend through the dorsal root ganglion. These two systems inform the cord of the neuromuscular activity being accomplished in regards to strength and duration.

rotation. Often the erector spinae muscles act on large segments they are not actually attached to. The forces that are expended also vary constantly, depending on the curvature of each functional unit and its direction of rotation. An example of one fascicle studied is shown in Figure 1-33 without major tissue deformity and neurological dysfunction.<sup>28</sup> In essence, the tissue deformation does not return to normal. Obviously the forces, duration, and frequency all play a role. The initial status of the tissue also determines the recovery.

## THE KINETIC SPINE

It appears that the kinetic spine, when violated, is the commonest cause of low back pain. This apparently occurs when a faulty kinetic action causes mechanical irritation of a nociceptor tissue. A review of normal kinetics of the spine is warranted.

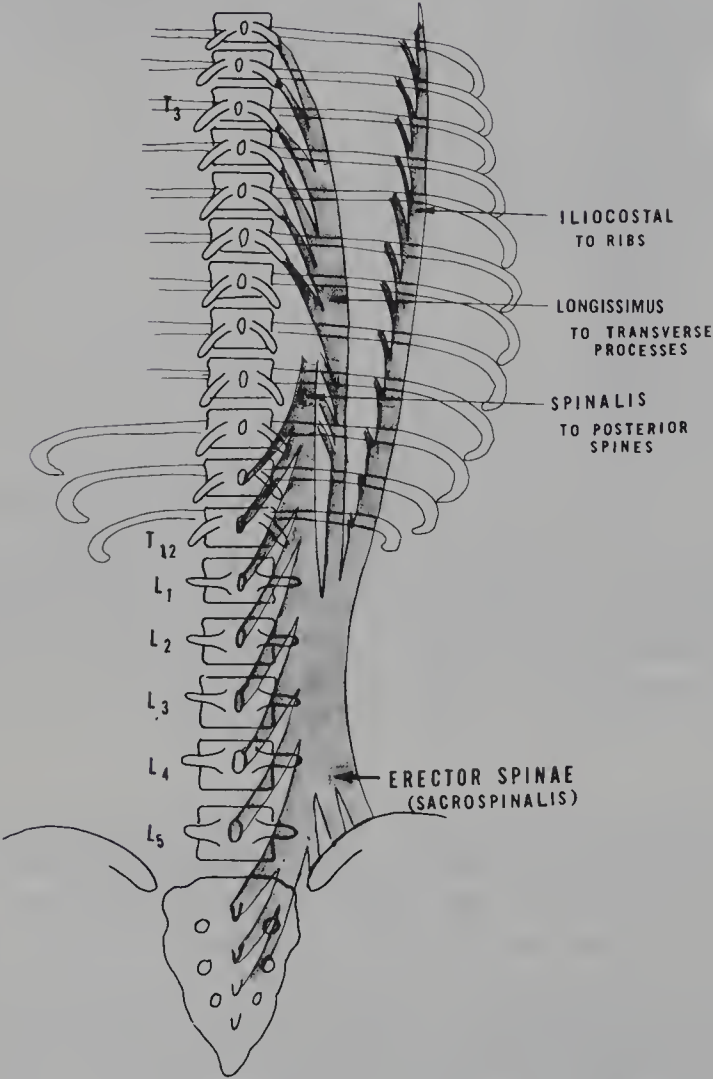


Figure 1-32. The extensor muscles of the back. The extensor muscles of the back and their origin and attachment are depicted.

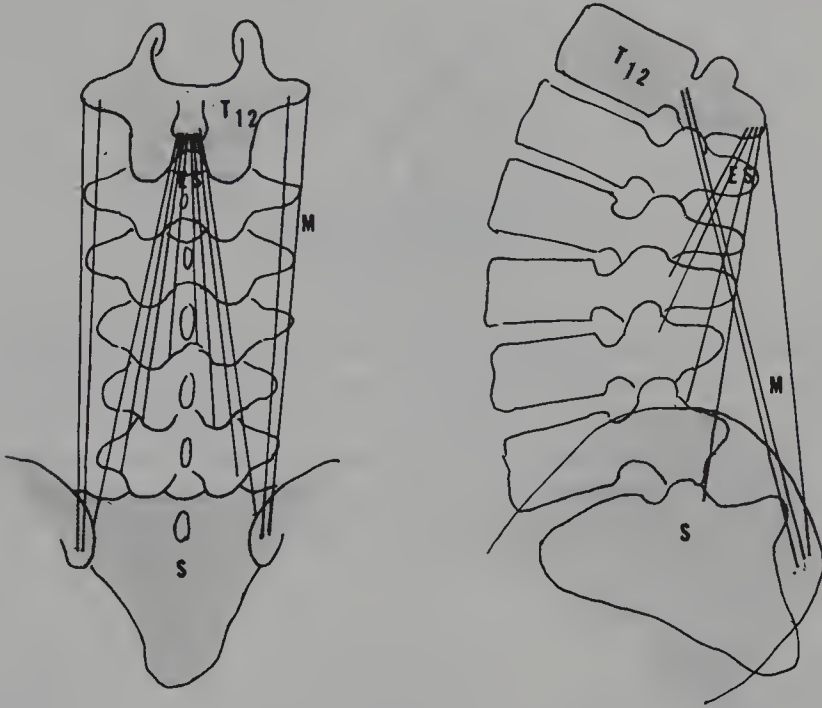


Figure 1-33. Posteroanterior and lateral views of the attachments of erector spinae and multifidus muscles. (Adapted from Bogduk, A, MacIntosh, JE, and Percy, MJ: Universal model of the lumbar back muscles in the upright position. *Spine* 17:897-913, 1992.)

The physiological erect static spine has a silent (inactive) erector spinae musculature: the passive subsystem of the spine.<sup>28</sup> Immediately upon intending forward or lateral flexion away from the exact center of gravity, initiating the kinetic spine, several neurophysiological factors initiate the active subsystem. The spindle system of the erector spinae musculature becomes activated (Figs. 1-34 and 1-35). The extrafusal fibers eccentrically contract and gradually elongate. The pattern of this neuromuscular action occurs centrally in the cerebral cortex and is moderated in the cord (Figs. 1-36 and 1-37).

Abnormal dynamic control of the lumbar paraspinal musculature, causing erratic lumbar-pelvic rhythm has been postulated as the cause of much low back pain. Persistence of this abnormal dynamic neurological regulation has been postulated as being responsible for chronic and recurrent low back pain.<sup>29</sup>

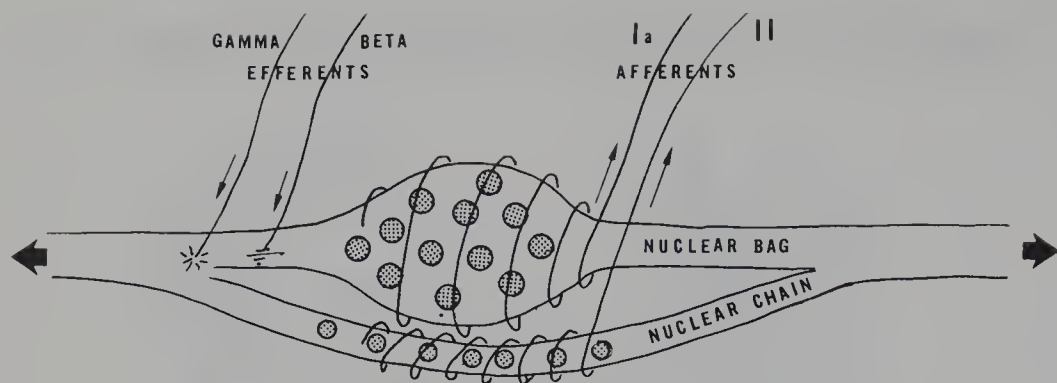


Figure 1-34. Intrafusal muscle spindle. The intrafusal spindle system has motor fibers through the gamma and beta efferent that control the length of the spindle. The sensory feedback from the spindle is transmitted via the Ia and II afferent fibers. As the fiber is lengthened (arrows) the system is stimulated by changes in the bag and the chain. The sensory impulses indicate the force, speed, and duration of the elongation. The spindle system is reset by the motor fibers for the next muscular extrafusal fiber contraction.

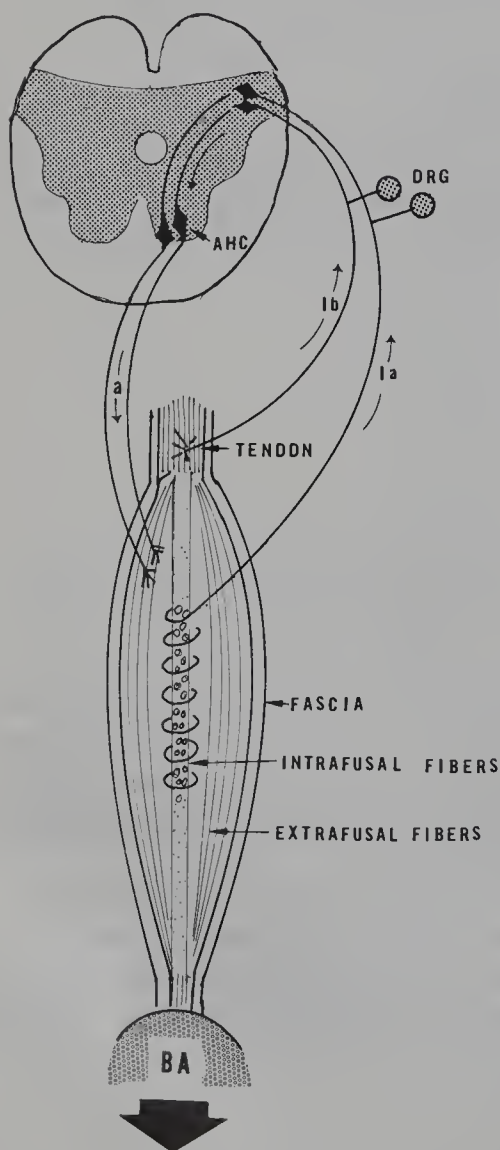


Figure 1-35. Spindle system function. The spindle system (intrafusal fibers) lays parallel to the extrafusal muscle fibers. When stretched they signal the cord (see Fig. 1-34) by way of the Ia fibers from the spindle and the Ib fibers from the Golgi tendon through the dorsal root ganglion (DRG). The impulses received in the gray matter have an internuncial connection with the contiguous anterior horn cells (AHC) that motor (a fiber) the extrafusal muscle fibers, causing an appropriate muscular contraction. The fascia elongates to its physiologic limit. The muscle tendon is connected to its bony attachment of the vertebral bodies (BA).

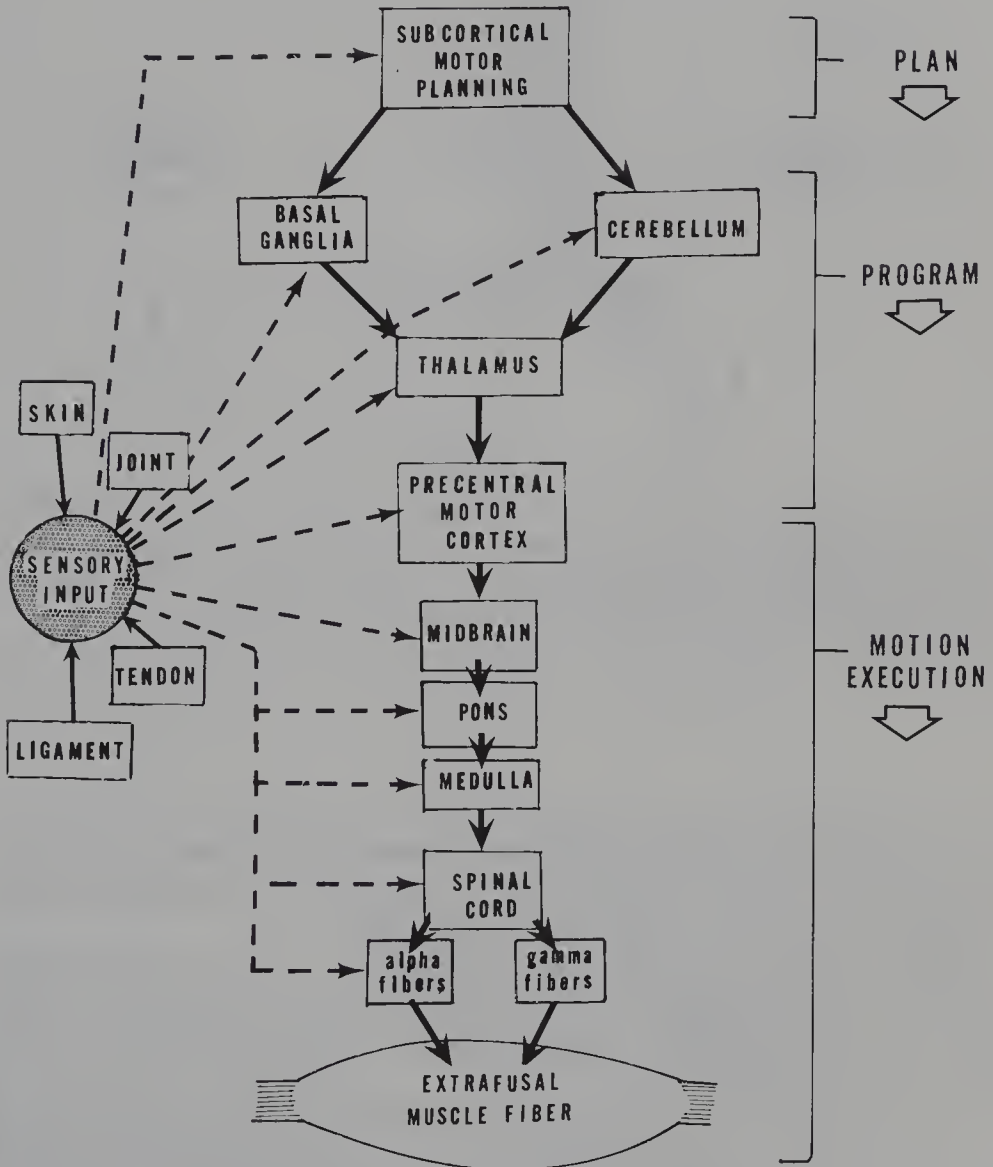


Figure 1-36. Spinal and supraspinal motor centers. The spinal and supraspinal motor centers show the planning, program, and motor execution from the subcortical motor center to the extrafusal muscle fiber. The sensory input is indicated.

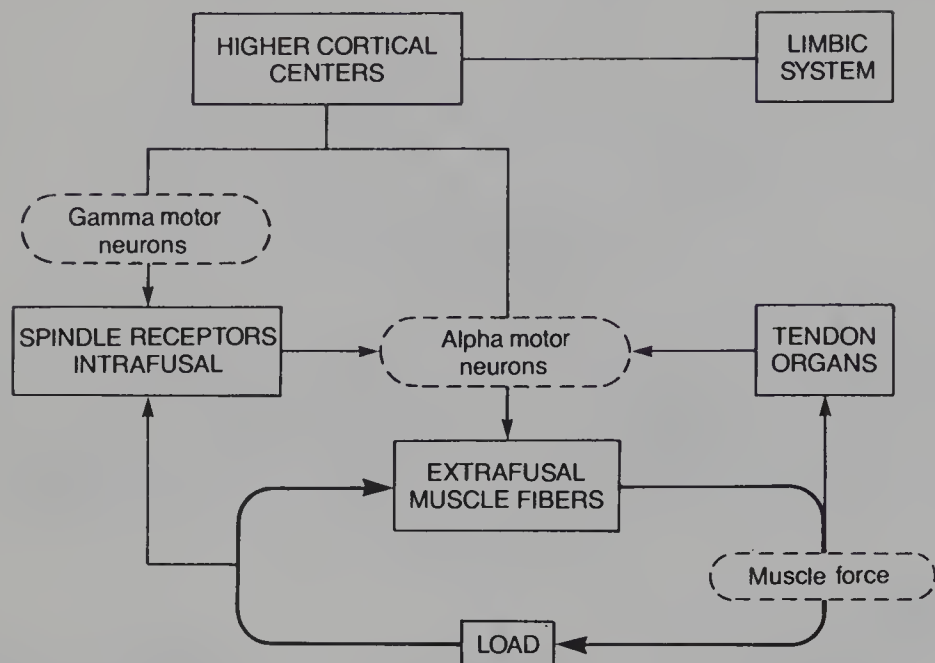


Figure 1-37. Higher cortical control of the spindle system. The higher cortical centers indicate the intended muscular activity to perform a specific action. A feedback system correlates this activity (see Fig. 1-36). The load expected from the intended activity determines the effort expended.

As the spine flexes, the erector spinae muscles gradually elongate at each functional unit (Fig. 1-38) in a controlled eccentric contraction.

The fascia elongates passively according to its original elasticity (Fig. 1-39), which is a collagen configuration.

During flexion there is also an elongation of the extraspinal ligaments, the posterior longitudinal ligament and the posterior annular fibers of the disk (Fig. 1-40). The nucleus of the disk deforms to its physiological limits.

During flexion, significant lateral motion, rotary motion, or both types of motion probably occurs<sup>19</sup> (Fig. 1-41). The facet alignment, normally parallel in the erect posture, deviates with approximation on the concave side and separation on the convex side (Fig. 1-42).

The lumbar spine is subjected to torsion (axial rotation) during many daily activities, and torsion is an important etiologic factor in low back pain and in disk damage and degeneration. The frequent history of a patient with low back pain that "the pain occurred on bending and twisting and returning to the erect position" is stated too often to be ignored.

The apophysial joints resist torsion, with Farfan<sup>30</sup> claiming 45% of rotational forces being limited by these facets and 10% by the inter- and



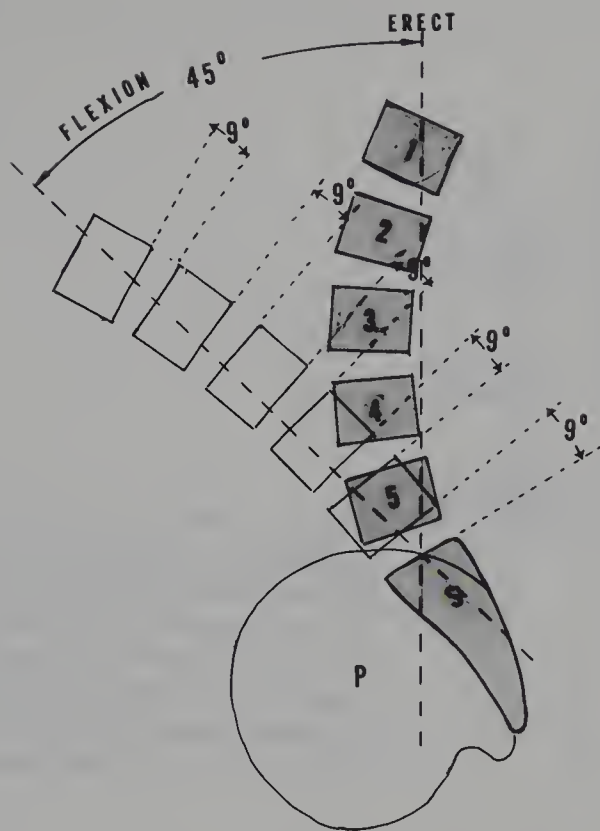


Figure 1-38. Total flexion pattern of lumbosacral spine. Each functional unit flexes approximately 8 to 10 degrees, approximating 9 degrees at each lumbar level. Because there are usually five lumbar vertebrae, flexion occurs to 45 degrees without the pelvis (P) rotating around the hip joint.

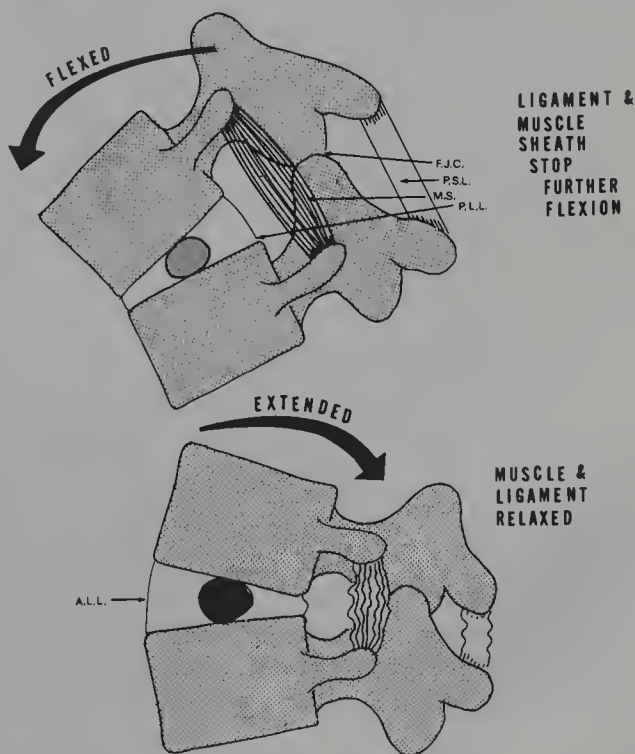


Figure 1-39. Restraint of flexion-extension of the lumbar functional unit. Excessive flexion is limited by the posterior tissues including the posterior longitudinal ligament (PLL), the sheath of the erector spinae muscles (MS), the posterior superior ligament (PSL), and the facet joint capsules (FJC). Extension is limited by the mechanical impact of the facet joints and the anterior longitudinal ligament (ALL).

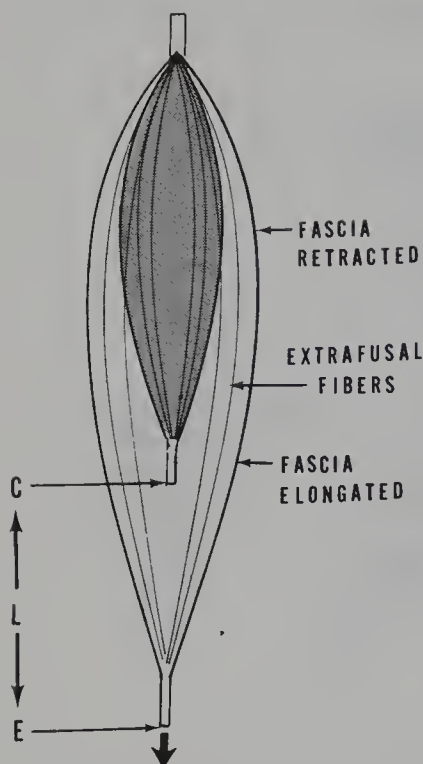


Figure 1-40. Fascial limits to muscular elongation. The fascia that encloses the extrafusal muscle fibers has its own elasticity. When the muscle is fully shortened (C) the fascia contracts. When the muscle is fully elongated the fascia elongates (E) to its physiologic limit. The *short arrow* represents the force elongating the muscle.

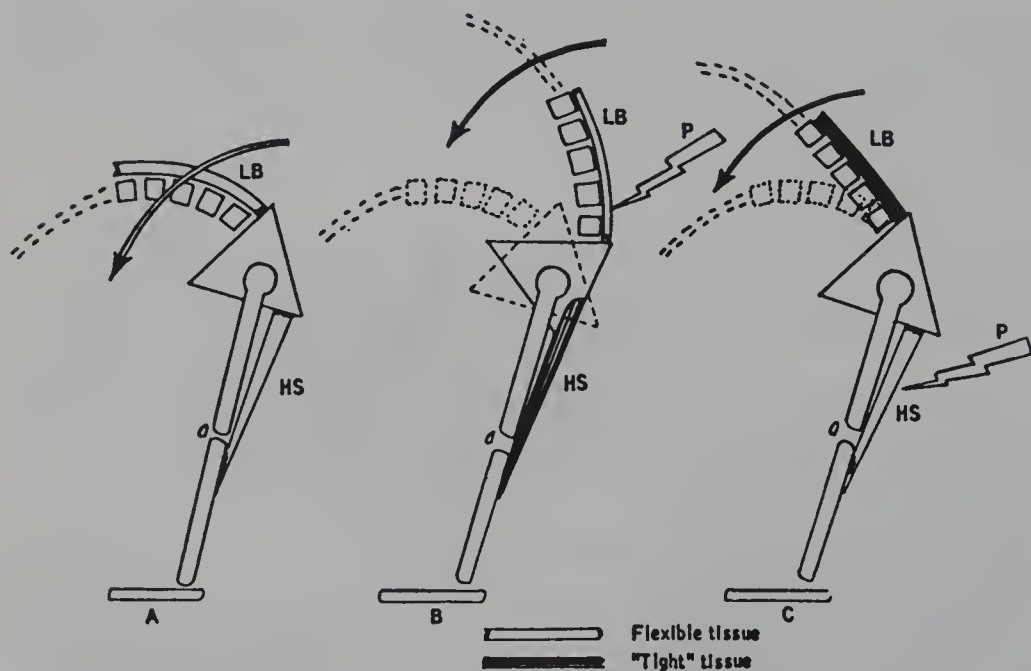


Figure 1-41. Mechanism of stretch pain in the tight hamstring and the tight low back syndromes. Normal flexibility with unrestricted lumbar-pelvic rhythm (A). Tight hamstrings (HS) restricting pelvic rotation and thereby causing excessive stretch of low back (LB) resulting in pain (P) (B). Tight low back (LB) performing an incomplete lumbar reversal and thus, by placing excessive stretch on the hamstrings (HS), causes pain (P) in both the hamstrings and the low back as well as a disrupted lumbar-pelvic rhythm (C).

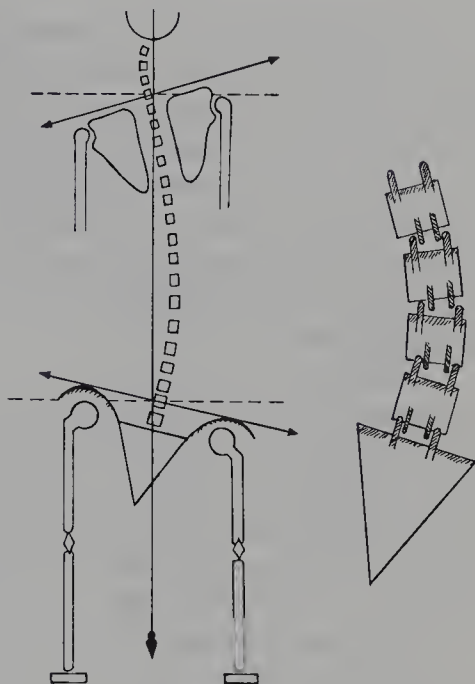


Figure 1-42. Facet asymmetry in lateral trunk flexion. Scoliosis secondary to pelvic obliquity (*left*) causes asymmetry of the facet alignment (*right*). This asymmetry predisposes impingement in erratic flexion and reextension of the lumbosacral spine.

supraspinous ligaments.<sup>31</sup> Early studies that axial rotation decreases as the spine flexes have been refuted. Flexion allegedly opens the facet joint space and permits greater rotation.

Many studies on single joint motion failed to include rotation and especially rotation during varying degrees of flexion. Shultz et al.<sup>32</sup> found increased rotation after removal of the posterior elements. Adams and Hutton<sup>11</sup> found increased flexion after incremental section of the supra- and interspinous ligaments, the ligamentous flavum and the facet capsules indicating resistance apparently coming from the facet capsules and the posterior annular fibers of the disk. Other studies showed that the greatest rotation occurred after removal of both facets and partial destruction of the disk annulus.<sup>28</sup>

A summarization concluded that the apophysial joint capsules resist rotation but not necessarily related to combined flexion.<sup>28</sup> The supra- and interspinous ligaments were considered less important in restricting rotation. The posterior longitudinal ligament and annular disk fibers were considered as important in restricting rotation. The addition of flexion definitely stretches the posterior annular fibers as noted by dynamic myelographic radiologic studies. The conclusion is that the posterior capsuloligamentous structures protect the disk annular tissues in rotation as do the erector spinae muscles, but the exact mechanism by which the annular fibers are torn remains unknown.

The role of trunk muscles aiding or enforcing spinal stability and their role in low back pain currently remain obscure in spite of many studies.<sup>33-38</sup> Without spinal muscles the spine has proven to be extremely unstable, buckling at critical loads of only 20N.<sup>39</sup>

The intraabdominal and intrathoracic cavity forces are the supporting structures of the vertebral spine and thus the strength of the extensors and the abdominal flexors are involved.<sup>36,40</sup> As stated in Chapter 6, the precise determination of muscle strength and endurance is still experimental.

Amplifying the concept expounded on the neurophysiological mechanisms<sup>41</sup> of the kinetic lumbosacral spine, let us note that the erector spinae muscles decelerate the trunk flexion and rotation before they accelerate extension and derotation in resuming the erect posture (Fig. 1-43). There is also a musculoligamentous component (Fig. 1-44) to these motions that will be clarified, as will the lumbar-pelvic rhythm of this action (Fig. 1-45).

Because the trunk muscles decelerate trunk flexion, this type of contraction must be clarified. There are three different types of muscle contraction: isometric, concentric, and eccentric.<sup>42-46</sup> In isometric con-

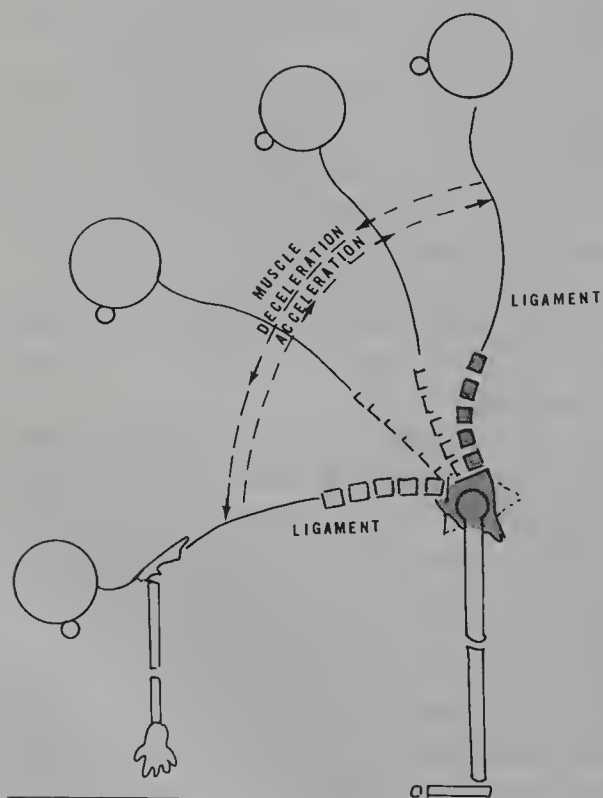


Figure 1-43. Muscular deceleration and acceleration of the flexing spine. From the erect stance the flexing spine flexes as the erector spinae muscles decelerate descent. When full flexion is achieved the muscles relax and support now relies exclusively on ligamentous and fascial support.

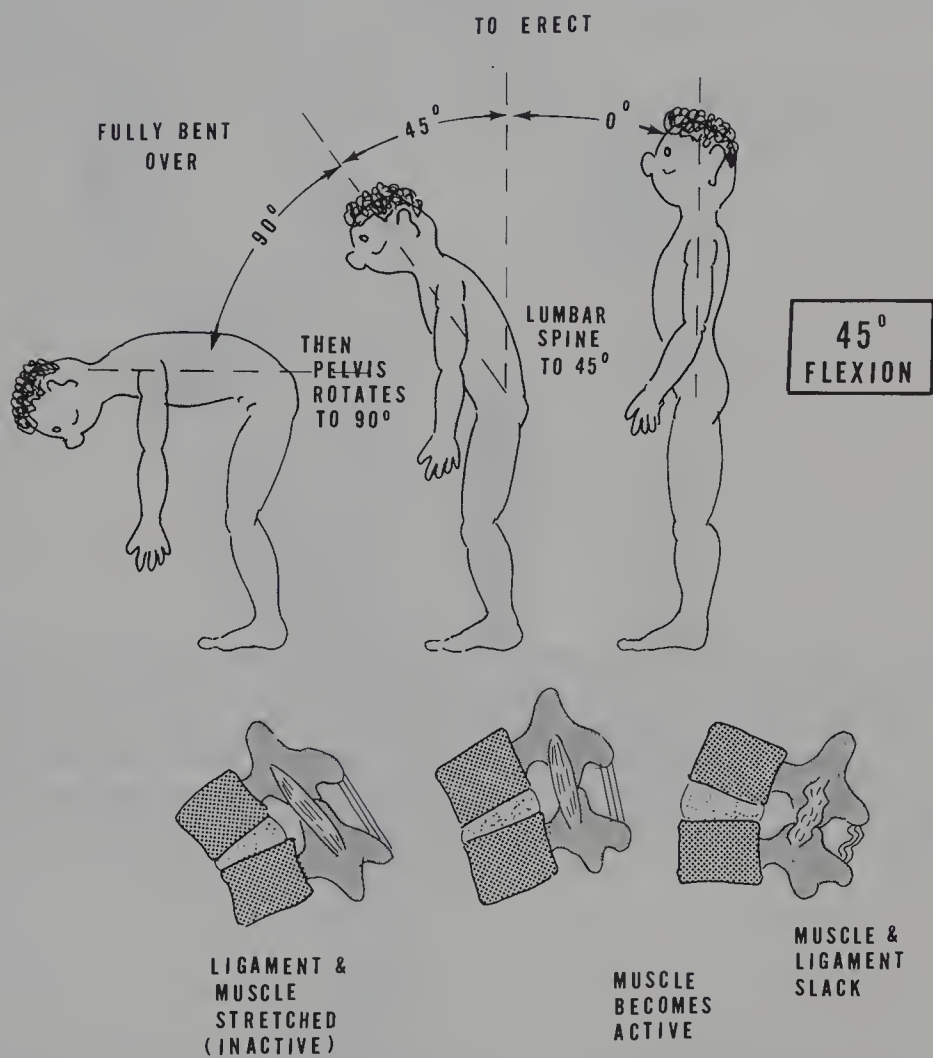


Figure 1-44. Muscular and ligament control in flexion reextension. The lumbar pelvic rhythm of flexion-extension is depicted with the fully erect posture at 0 degrees. The right lower figure reveals the muscles and ligaments being slack. As the person flexes to 45 degrees flexion occurs mostly in the lumbar spine with the muscles decelerating and the pelvis remaining static. After 45 degrees of flexion the pelvis rotates as does continued trunk flexion until all the muscles become inactive but tension occurs in the ligaments and the fascia.



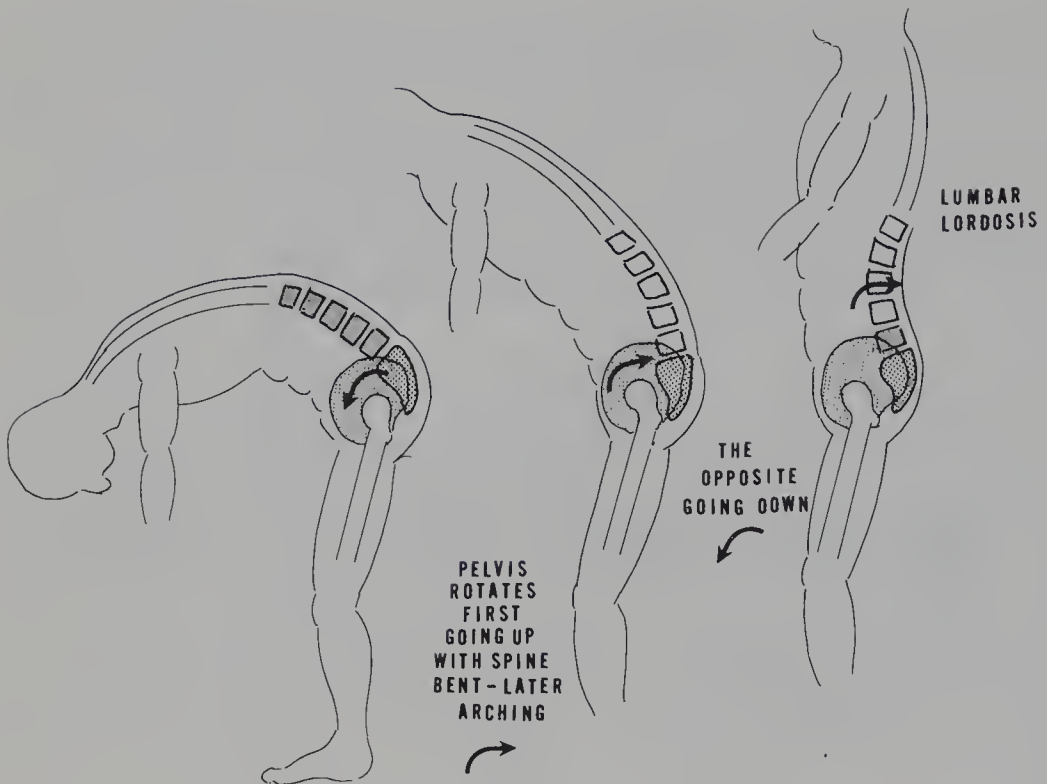


Figure 1-45. Lumbar-pelvic rhythm in flexion-reextension of the lumbosacral spine. The lumbar spine pelvic rhythm in flexion and reextension has been stated in Figure 1-44. Once the trunk is fully flexed the pelvis is fully rotated and the spine fully flexed. Return to the erect posture requires derotation of the pelvis first with the spine remaining flexed until midpoint (approximately 45 degrees) then the pelvis slows in derotation and the spine regains a slow lordosis.

traction the length of the muscle does not change. In concentric contraction the muscle shortens as the forces are expended. Eccentric contraction occurs when the external force is greater than the internal force of the muscles.<sup>47</sup> Eccentric contraction is considered as negative work, in which, as the muscle lengthens, it is contracted and stretches the noncontractile tissue. The internal muscle tension is thus a combination of both the contractile and noncontractile tissues.<sup>48</sup> Of the three types of muscle contraction eccentric contraction creates the highest muscle tension yet the lowest energy consumption.<sup>43</sup> In spite of a significant role of eccentric muscle contraction in low back function, its role in axial torque rotation and causation of symptomatic patients remains conjectural.



Motion of the spine occurs in varying directions and combinations thereof. The large erector spinae muscle fascicles extend the spine but also have a rotational effect (Fig. 1 – 46). There are also smaller intrinsic muscles that rotate the segments (see Fig. 1 – 32). Measurement of rotation range of motion has been difficult to document as it differs with different degrees of flexion. The tissues that resist rotation within the functional units are difficult to identify.

In the upright lordotic posture the lumbar intervertebral joint has an axial rotational range of 2° to 3°. If this range is exceeded, the

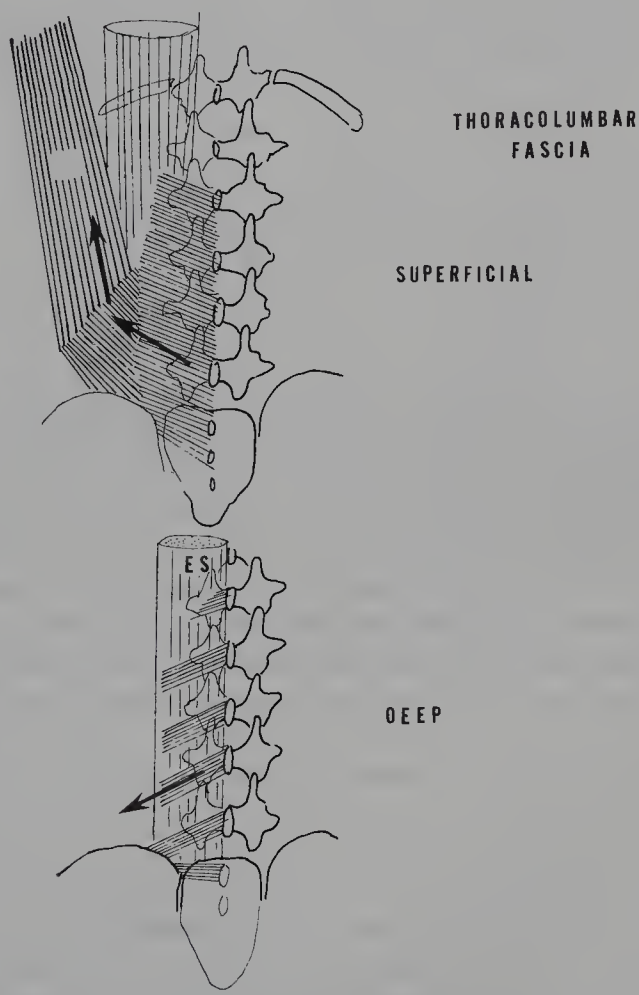


Figure 1 – 46. Lines of pull of the thoracolumbar fascia layers. The three layers of the fascia act in three different directions of tension. The superficial layers attach from the posterior superior spines at the midline. Medially their lines of stretch are lateral with a slight cervical direction. The raphe into the latissimus dorsi (LD) changes the direction of tension cephalad making the tension in the medial fibers greater. The deep layers of the fascia contain the erector spinae muscles (ES) and their pull is in a lateral and caudal direction.

movement is initially resisted by the facet joints. The annular fibers permit a greater degree of rotation so if any tearing occurs it must be after exceeding the limits of the facets.<sup>49,50</sup>

In a flexed position, with a reversed lordosis, the facets separate, allowing greater axial rotation before there is facet blocking.<sup>51,52</sup> Rotational studies have usually been done in the seated position, thus making upright determination invalid.

Rotation occurs at the thoracolumbar segment and at the lumbar segments as the thoracic zygapophysial (facets) joints are in a sagittal alignment and allow no significant rotation.<sup>53</sup> Rotation in the standing flexed position differs from rotation in the seated position as in the former the erector spinae muscles must contract to sustain the flexed position and thus apply compressive forces on the functional units.

In the erect posture the lordosis causes a compression of the posterior annular fibers as well as approximation of the facets causing rotational restriction (Fig. 1-20). The anterior annular fibers are under tension and lateral shear forces resist rotation.

In the flexed position the facets separate and the posterior annular fibers are placed under tension, resisting rotation. Excessive rotational forces now are no longer resisted by the facets but by the posterior annular fibers. The excessive rotation permitted in this position (approximately 2°)<sup>13</sup> can now tear the posterior annular fibers.<sup>54</sup>

As previously stated, the lengths of the annular fibers vary according to their distance from the center of the disk, thus causing a different tearing. The more central fibers are shorter; hence, they have less elongation potential and tear first on rotational forces.<sup>55</sup>

The compressibility of the nucleus and the variable extensibility of the annular fibers allow flexion-extension and some rotational motion. These degrees of mobility have been studied and standardized. Compression forces merely decrease the angulation of the annular fibers without elongating the individual fibers to any significant degree. Flexion and extension change the angulation of the fibers and cause some degree of elongation of these fibers. Translation of the functional units, which occurs on flexion, increases this elongation (Fig. 1-47).

Mobility of the spine has been studied radiologically both in a static as well as kinetic aspect,<sup>56-61</sup> yet little is known or universally accepted as the normal range of motion. In spite of this ignorance, the medical model continues to measure range of motion and attribute significance to its variations. Treatment protocols also aim at increasing or decreasing the range of motion as being pertinent to the causation of pain and dysfunction.

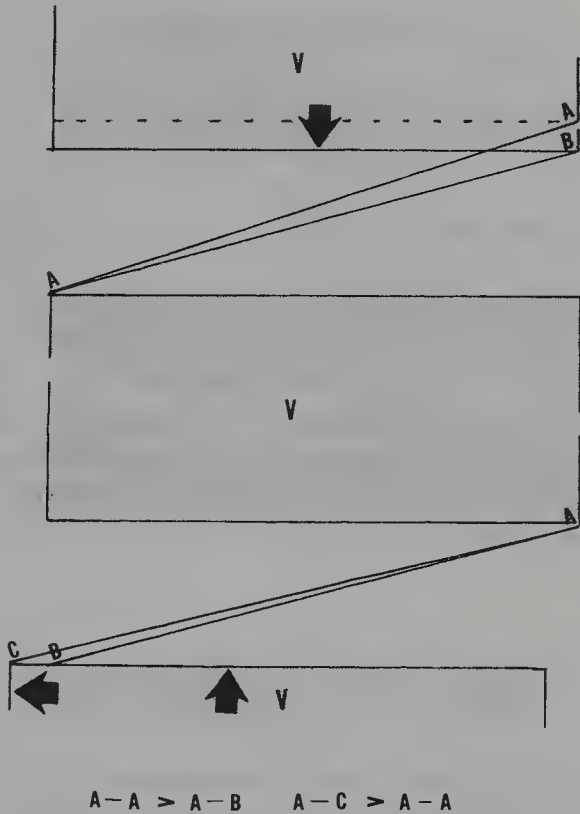


Figure 1-47. Collagen length from disk compression and translation. The top of the figure shows the difference in the length of the collagen fibers (A-A) when the vertebrae are apart (*dotted line*) and when compressed (*arrow*). It is apparent that the fibers are shorter when the disk is compressed ( $A-A > A-B$ ). When there is compression (*vertical arrow*) and translation (*horizontal arrow*) the collagen fibers are at their full elongation ( $A-C > A-A$ ). Physiologically the collagen fibers adopt this elongation from compression and shear. If either force is excessive the collagen fibers can tear. The internal collagen fibers near the nucleus (not shown in this figure) have less flexibility.

Where and to what degree motion occurs within a spinal functional unit remains obscure. Lateral flexion, with or without rotation, cannot be objectively measured nor are the measurements obtained consistent or necessarily related to symptomatology (Fig. 1-48). The influence of age and gender on spinal mobility remains unclear.

Acute inflammation from trauma causes immediate limitation. Fear of pain recurrence or aggravation of already existing pain also decreases

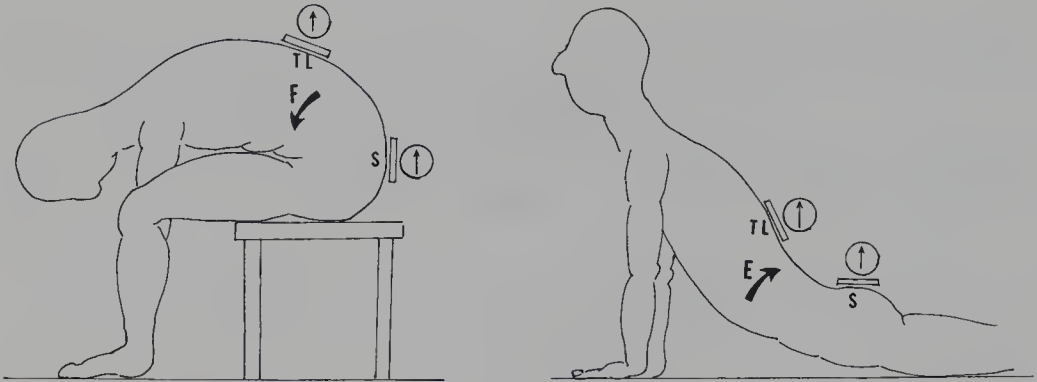


Figure 1-48. Measurement of lumbar spinal range of motion. A fluid-filled inclinometer (MEDesign, Southport, UK) is placed on the thoracolumbar spine (TL) and on the sacrum (S). During flexion (F) the difference in angle is determined as it is during extension (E).

range of motion for a limited time. Persistence of pain or the incurred fear of pain aggravates the restriction until structural changes occur in the dense connective tissues (muscles, fascia, and ligaments).<sup>58</sup>

Translation occurs during flexion, extension, and rotation (Fig. 1-49). There is a difference of motion, translatory or angular, between various levels (L1, L2, L3, L4, L5, and S1) in asymptomatic people<sup>62</sup> that varies from 3 mm to 10° to 20°.<sup>58</sup> These minor differences add to the difficulty of objective documentation.

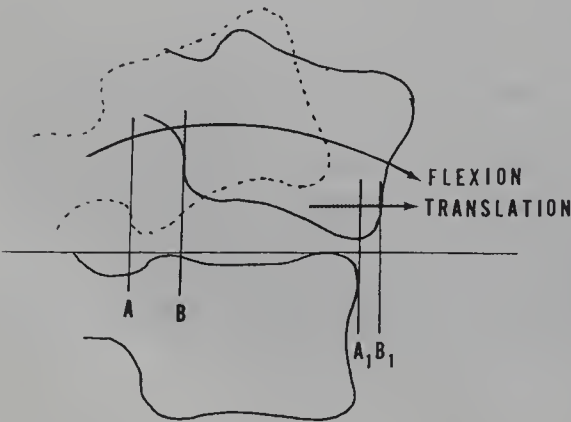


Figure 1-49. Translation within a functional unit. As there is flexion within a functional unit the superior vertebra flexes and translates anteriorly from A-B and anteriorly A1-B1. The intervertebral disk narrows anteriorly and the pedicles separate to increase the diameter of the foramen.

SPINAL STABILITY

Movement of spinal segments has presented a clinical problem of stability as<sup>63,64</sup> being within normal limits and possibly symptomatic. A neutral zone of motion has been postulated but not clearly defined.

A spinal stabilizing system consisting of three subsystems has been postulated.<sup>65</sup> The neural subsystem initiates precise movement and coordinates all motion factors. The disks, facet articulations, and their capsules and ligaments are the passive subsystem, and the muscles and their tendons are the active subsystem (Fig. 1–50).

Within the neural subsystem are the nerve components, innervating the muscles, and the sensory components, including the spindle and Golgi systems providing the needed coordination. The subsystem determines the rate of motion and the force.

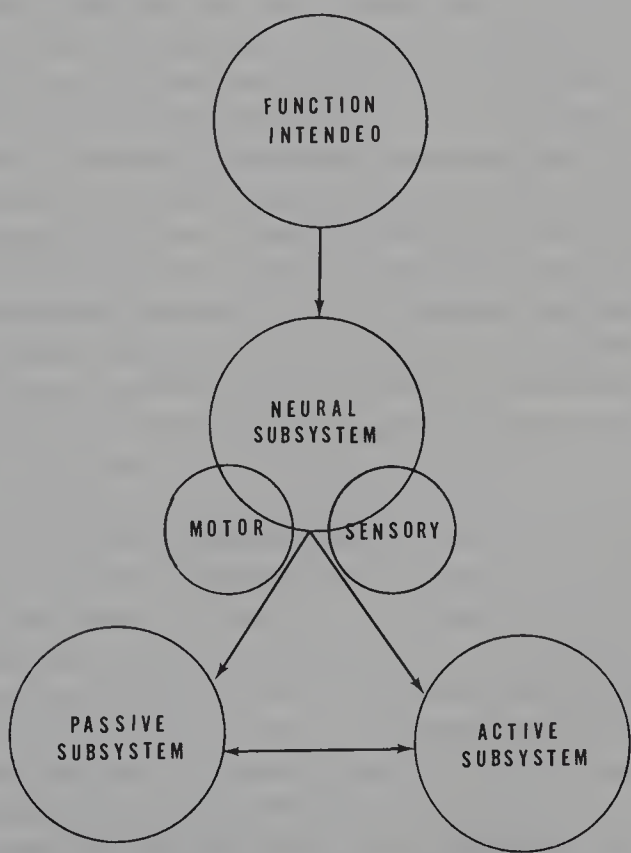


Figure 1–50. Spinal stability system. Spinal stability is effected via three subsystems acting in coordination. The neural subsystem initiates and coordinates both other systems through motor and sensory components. The stabilization subsystems are the passive vertebral column components of disk, ligaments, and joint capsule whereas the active subsystem comprises the muscles of the column.



The basic biomechanical functions of the spinal systems are to allow appropriate movement to accomplish the intended action, to allow appropriate movement to carry the load with commensurate muscular efforts, and to protect the involved tissues from injurious forces and stresses.

## LUMBAR ERECTOR SPINAE MUSCLES

The lumbar erector spinae muscles play a vital role in maintenance of the static spine and are especially involved in the kinetic spine. The lumbar extensor spinae muscles lie lateral to the multifidus and extend from the pelvis into the thoracic spine. This group of muscles is not a common muscle mass but rather consists of four divisions: the longissimus thoracis pars thoracis, the iliocostalis lumborum pars thoracis, the longissimus thoracis pars lumborum, and the iliocostalis lumborum pars lumborum, so designated depending on their origin and attachments.<sup>65</sup>

The two thoracic divisions arise from the transverse processes of the thoracic vertebrae and the ribs to terminate in an aponeurosis, and the two lumbar divisions arise from the transverse processes of the lumbar vertebrae and insert directly on the ilium without connection to the aponeurosis. The aponeurosis (fascia), is also a functional tissue in low back activities. As the four divisions assume different sites of attachment they must also have different functions on the lumbosacral spine.

As the fascicles of the lumbar erector spinae muscle group have different orientations, they exert different posterior and lateral shear and compression at the functional units. In the erect posture the thoracic fibrils exert compression at all levels, whereas the lumbar fascicles exert compression only at the upper lumbar segments.

The muscle actions of the back muscles must therefore be ascertained from their attachments and alignment. The multifidus muscle fibers are oriented at right angles to the transverse plane; therefore, they cannot exert posterior translation of the functional unit.<sup>66</sup> The multifidus muscle fibers can only produce posteriosagittal rotation or oppose anterosagittal rotation.

The lumbar fibers of the longissimus thoracis and the iliocostalis lumborum incline downward and posteriorly from their transverse process attachments; therefore, they exert posterior and sagittal rotation and posterior translation. The lumbar fibers also minimize or prevent forward translation.

In essence, the multifidus and the lumbar fibers of the erector groups are extensors and control deceleration of forward flexion. Their function in rotation remains obscure. Unilaterally they cause lateral



flexion. Gravity is the major force on the spine in flexion and the erector spinae muscles act eccentrically to decelerate until full flexion has been reached and reextension is expected.

## MUSCLES IN SPINAL FUNCTION

Many studies have been directed at evaluating the isometric and concentric contraction of the low back muscles, but few have elucidated the effect and documentation of eccentric muscle contraction.<sup>65-69</sup> The eccentric contraction of the extensor spinae muscles is predominant in most back activities of bending and ultimate lifting and should be addressed.

The position of the patient during testing has also varied from prone, supine, sitting, and standing, with and without pelvic immobilization to exclude the forces of the lower extremities. With any of these constraints a true evaluation has been difficult if even indicative.

In testing for eccentric versus concentric and isometric strength of patients with or without low back pain, the same findings were apparent: there was discrepancy between flexors and extensors but no significant difference between symptomatic and asymptomatic subjects. Eccentric contractions were considered to be conducive to muscular damage during testing even when velocity was controlled.

The muscle mass being evaluated in most of these studies has been the erector spinae muscles, which traditionally arise from the lumbosacral region to insert into lumbar and thoracic transverse processes and the ribs. Their actions have been those of extension and, for the more lateral fibers, lateral flexion.<sup>70</sup>

More recently the erector mass has been depicted as consisting of four divisions: longissimus thoracis pars thoracis, iliocostalis lumborum pars thoracis, longissimus thoracis pars lumborum, and iliocostalis lumborum pars lumborum.<sup>71</sup> The two thoracic divisions arise from the transverse processes and ribs forming the erector spinae aponeurosis in the lumbar region. The two lumbar divisions arise from the transverse processes of the lumbar vertebrae and insert into the ilium. These four divisions are considered to have different actions by virtue of their attachments (Figs. 1-51 and 1-24).

Because the sites of attachment do not vary regardless of vertebrae size or fascicle length, their action is constant. The specific action of the attachments can be modeled and computed. Their action, as is the action of the multifidus, is across numerous sections of the spinal column and varies as do the changes in the lumbar lordosis or kyphosis.

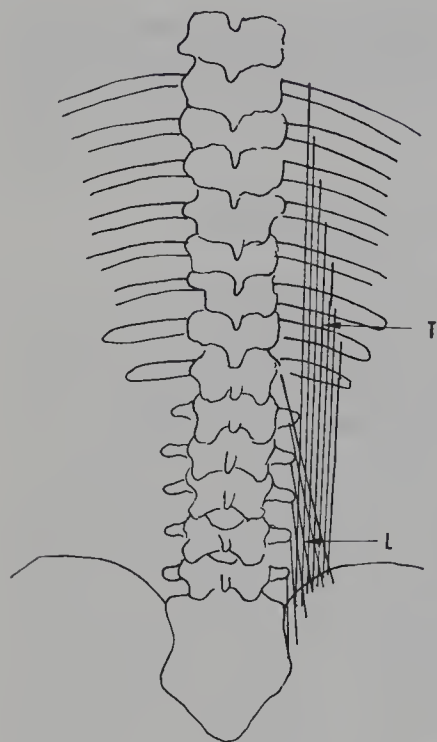


Figure 1-51. Attachments of the lumbar erector spinae muscles. The attachments and insertions of the erector spinae muscles are depicted with *T* being the thoracis group and *L* the lumborum group. By evaluating each muscle fascicle the action can be determined. There are four divisions: longissimus thoracis pars thoracic, iliocostalis lumborum pars thoracis, longissimus thoracis pars lumborum, and iliocostalis lumborum pars lumborum. The first two (thoracis) arise from the thoracic transverse process of the ribs and the latter two (lumborum) from the transverse processes of the lumbar vertebrae.

How the erector spinae muscles function as a subsystem in the spinal stability system needs some clarification. When flexing the trunk the erector spinae muscles elongate and shorten in reextension. They also exert compressive forces on the functional units.

The configuration, direction, and angulation of the fibrils differ in the erect posture and in flexion.<sup>72,73</sup> In the erect posture, the angulation of the fibrils varies according to the degree of lordosis and the distance from their axes of rotation as well as in each functional unit. In flexion, this alignment changes with every degree of angulation (flexion) of each functional unit (Fig. 1-24), thus it changes as the spine progressively flexes.

In flexion there is a significant elongation of the erector spinae fibrils and changes in their orientation to the spine. As the multifidus and the iliocostalis-longissimus crisscross during flexion, they vary in their alignments and essentially do not change in their torque or compressive forces. The elongation of the fibrils (15% to 59%) reduces their active tension but increases their passive tension.

In the erect posture the fascicles of the multifidus muscles are oriented in a dorso-caudal direction but become ventro-caudal in flexion, whereas the fascicles of the longissimus merely align more to the longitudinal axis of the spine.

## Thoracolumbar Fascia

The thoracolumbar fascia encloses all the muscles of the erector spinae and quadratus lumborum muscles. It passively plays a major role in the fibroelastic support of the spine both in flexion and reextension. It exerts pull in conjunction with the erector spinae muscles, which can paradoxically be termed *active-passive pull* (see Fig. 1–47).

Muscle pain is recognized as a factor in low back pain. The mechanism is an increase in activity of the nociceptors within the muscle belies by substances such as bradykinin.<sup>17</sup> Normal muscular activity does not activate these nociceptors.

The location of these free nerve endings is in the wall of arterioles and connective tissue of muscle.<sup>74</sup> These free nerve endings are almost completely ensheathed by Schwann cells, with only small portions uncovered and exposed to the interstitial fluid.<sup>75</sup> Their exact morphology is unclear.

Neuropeptides are the substances that stimulate the nociceptor sites. No specific neuropeptide has been identified as the type that irritates muscle endings, but substance P is considered most likely.<sup>76</sup> The peptide patterns have been similar to those of cutaneous and visceral organs.<sup>77</sup> Many of these neuropeptides are found not only in nociceptive units but also within the spindles innervation so the conclusion is that many of these neuropeptides are found in thin myelinated and nonmyelinated muscle afferents irrespective of their sensory function.<sup>78</sup>

These substance P neuropeptides are released not only from the spinal terminals of the afferent fibers but also from the receptive endings in the periphery. They have a strong vascular action, thus they effect the environment after their release. The bulk of the substance P released in the dorsal ganglion is transported to the peripheral nerve ending<sup>79</sup> and is considered to initiate “neurogenic inflammation.”<sup>80</sup>

The exact mechanism by which these neuropeptides cause muscle pain is unclear. The probability exists that they cause a vasodilation in the muscle that is firmly enclosed within its fascia and increases the intramuscular pressure, resulting in pain.

The mechanical forces that act on the muscles, exciting their nociceptors, are from breaks in muscle fibers and extravasation within the muscle belly as well as the liberation of neuropeptides.

A muscle that is forced to perform physical work of unaccustomed intensity or duration undergoes histologic alterations, including necrosis. This is especially true after eccentric contraction.<sup>81–83</sup> As has been stated, eccentric contraction induces trauma as the external forces acting on the muscle are greater than those produced by the muscle.<sup>84</sup> This is assumed to occur as there are fewer muscle fibers acting during

eccentric contraction than in positive work of similar intensity and the mechanical elongation stress on the Z bands of the sarcomeres causes damage.<sup>85</sup>

Ischemia of the muscle has been postulated as a major cause of muscle pain. Merely interfering with the circulation of muscle does not cause pain,<sup>86</sup> but contraction of that muscle during ischemia does cause pain. What the mechanism is remains unclear. During ischemia a plasma protein (BKN)<sup>86</sup> is released that allegedly irritates the nociceptor endings;<sup>87</sup> however, that also remains unconfirmed.

Increased muscle tone as a factor in pain remains unclear albeit being considered as pertinent. *Muscle tone* remains undefined as a neuromuscular activity just as a relaxed muscle is electromyographically silent.<sup>88,89</sup> Tone has been equated as the result of viscoelasticity such as osmotic pressure from fluids, elastic tonus within the connective tissues, and pressure within fascial compartments rather than a neuromuscular tonus. It has been clinically determined that increased muscle tone can result from resistance or passive movement.

Pain that results from spasm or cramps remains unclear as the intramuscular pressure has not been found to be significantly elevated.<sup>90</sup> The neural mechanism postulated as occurring is the afferent nociceptive impulse synapsis with the alpha-motor neurons at the cord level (Figure 1-52).<sup>91-95</sup>

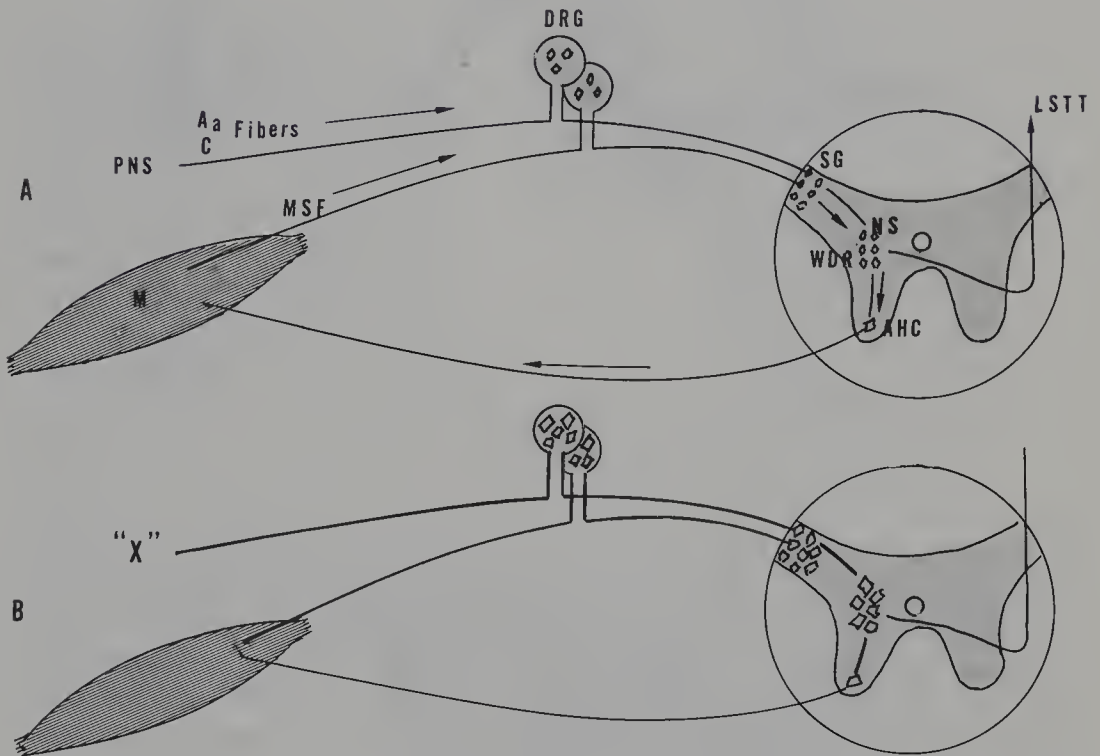
The erector spinae muscles that have been fully discussed cannot generate sufficient movement to lift objects exceeding 35 kg.<sup>105</sup> Thus, in lifting heavy objects, tissues other than the erector spinae muscles must be brought into play (see Fig. 1-45). The ligaments or fascia must be considered along with the major muscles groups being involved (Figs. 1-53 and 1-54).

It is believed that abdominal pressure relieves intradiskal pressure in any lifting activity. This concept envisions an "air bag" within the abdominal cavity (Fig. 1-55) that acts to decompress the pressure on the disks. This concept also includes intrathoracic pressure as well as intraabdominal pressure. (Fig. 1-56).

Intracompartmental pressure within the fascial sheaths (Fig. 1-57) is also considered a factor unloading the spine (Fig. 1-58) and explains the value of strong abdominal muscles, especially the obliques, which insert on the fascia of the erector spinae muscles. By their attachment to the fascia, they laterally elongate the fascia, making a stronger extensor component. They also stiffen the fascia, increasing the intracompartmental pressure.

The efficiency of the lifting process employing numerous tissues in addition to the erector spinae implies a synchrony of these associated tissues.





**Figure 1-52.** Cord neural synapses in muscle spasm. *A*, Impulses enter the gray matter of the cord from peripheral nociceptive sites (PNS) via Aa and C fibers, through the dorsal root ganglion (DRG) into the substantia gelatinosa (SG). An internuncial neuronal circuit activates the nociceptive neurones (NS), the wide dynamic range neurones (WDR) then transfers across the cord to ascend to the thalamic sites via the lateral spinothalamic tracts (LSTT). A neuronal tract connects the WDR to the anterior horn cells (AHC), which innervate the muscles of that specific segment. This conceivably is the segmental spasm that occurs to immobilize a painful peripheral site. Afferent nociceptive impulses originate from muscles that are in spasm or otherwise inflamed (MSF). *B*, Intense persistent peripheral inflammation (X) causes the neurones in the DRG, the SG, NS, and WDR to increase in size and number, conceivably causing greater pain and greater chronicity.

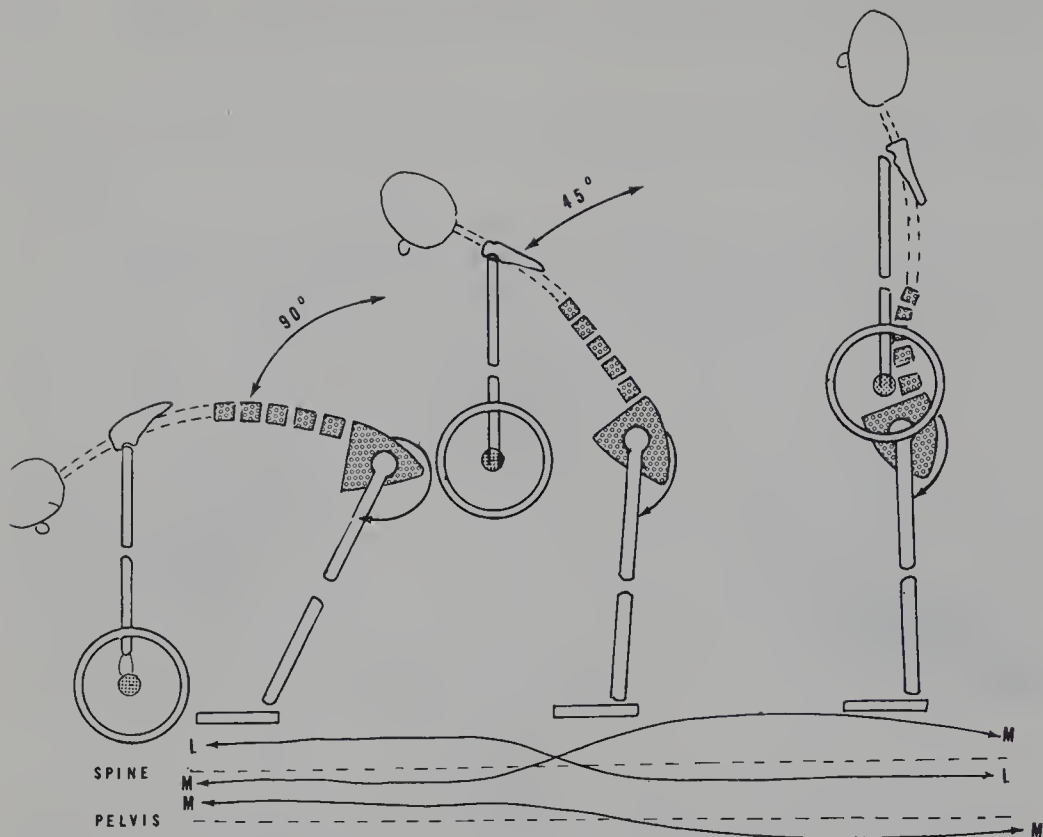


Figure 1-53. Muscular-ligamentous control in lifting. In lifting from the fully flexed lumbo-sacral spine (90 degrees) the spine is supported by its ligamentous and fascial structures (L). The muscular effort (M) is largely rotation of the buttocks (*curved arrow*). When the ascending spine reaches 45 degrees the erector spinae muscle has become the extensor forces with the ligaments decreasing their tension. The muscles continue to exert the needed force until the spine is fully erect.

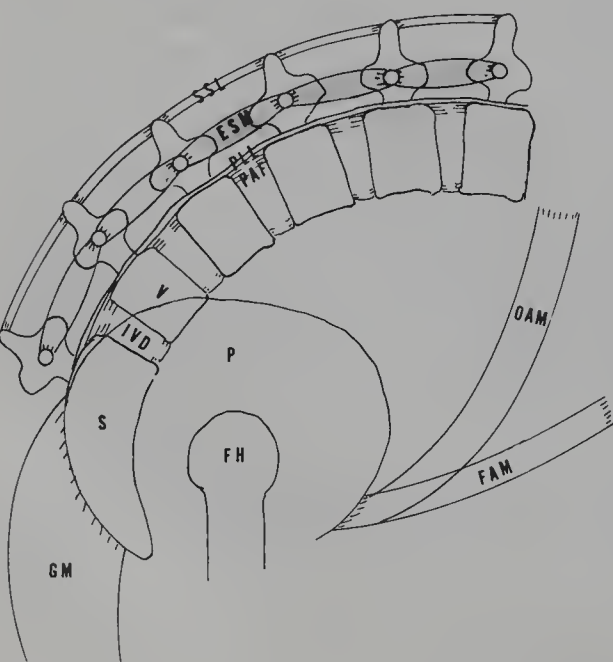


Figure 1-54. Structures and function of the lumbar spine. The flexed spine is depicted. The tissues involved are supraspinous ligament (SSL), erector spinae muscle (ESM), posterior longitudinal ligament (PLL), posterior anular fibers (PAF), vertebra (V), intervertebral disk (IVD), sacrum (S), gluteal muscles (GM), pelvis (P), femoral head (FH), oblique abdominal muscles (OAM), flexor abdominal muscles (FAM).



Figure 1-55. The air bag theory of unloading the spine. *A* denotes the pressure within the abdominal cavity and *B* within the erector muscular-fascial compartments. The intra-abdominal cavity arrows depict the pressure gradients that unload the spine.

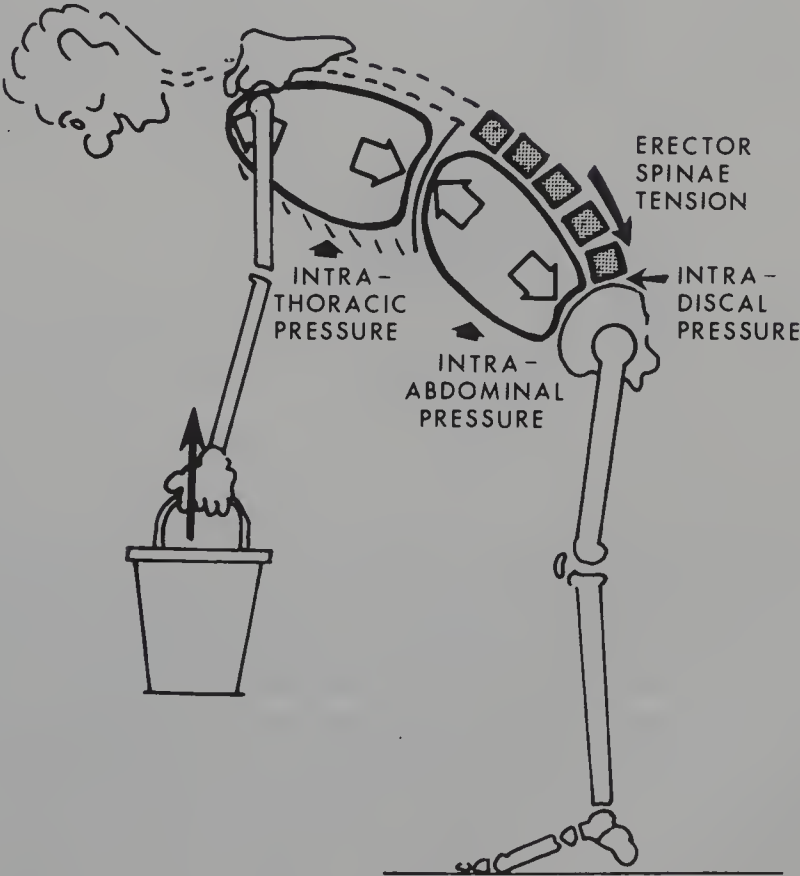
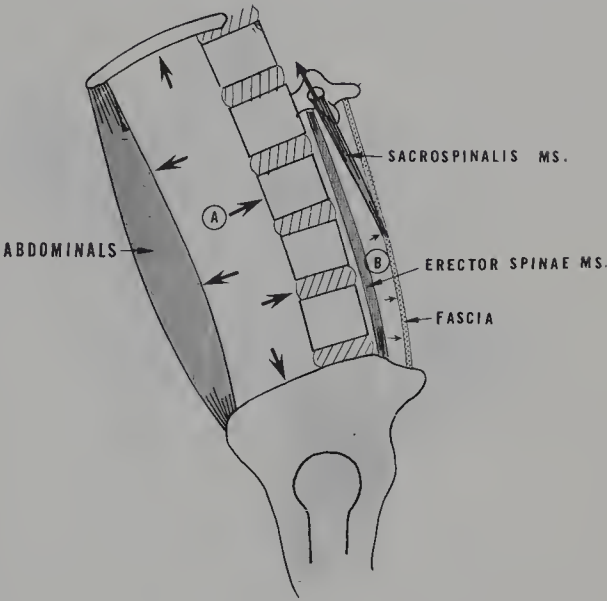


Figure 1-56. Abdominal-thoracic support of the spine. The internal pressure created within the thoracic and abdominal cavities during the act of lifting unload the intradiscal pressure within the intervertebral disks.

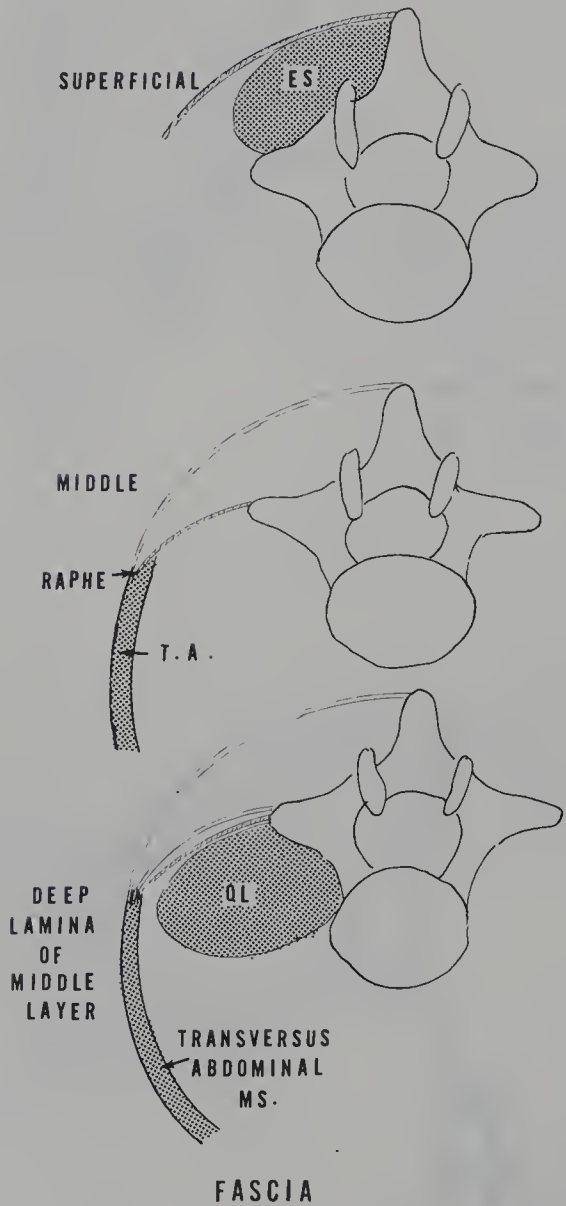
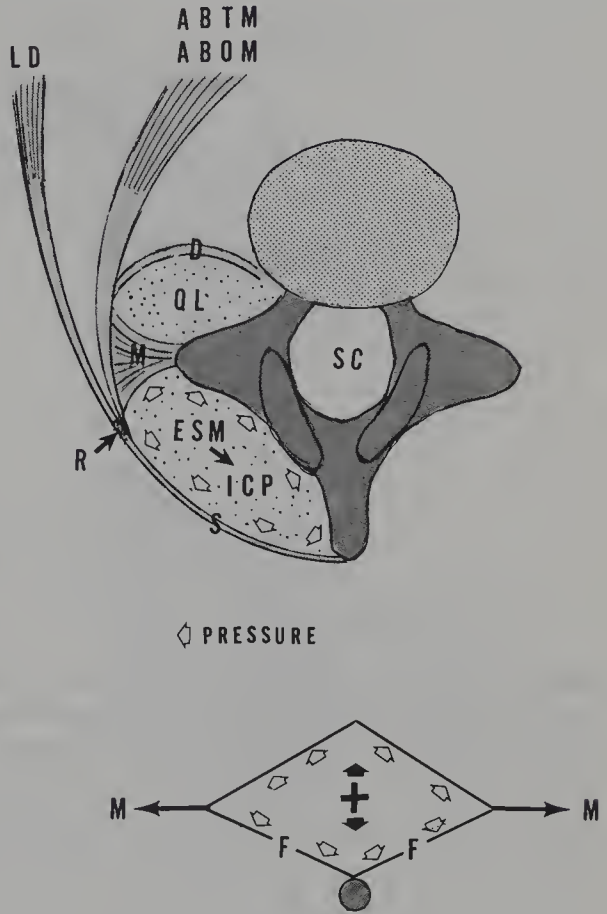


Figure 1 - 57. Thoracolumbar fascia. This fascia has three layers. The superficial covers all the erector spinae muscles (ES) but does not attach to the transverse abdominal muscles. The middle layer proceeds from the transverse abdominal muscles (TA) forms a raphe for which two layers of fascia proceed to attach to the vertebral transverse processes and superior spines enclosing the erector muscle into a compartment. The deep layer also proceeds from the raphe with the deep lamina of the deep layer enclosing the quadratus lumborum muscle (QL).

Figure 1-58. Intracompartmental pressure within fascial sheaths of back muscles. The fascial sheaths (see Fig. 1-56) emanate from the latissimus dorsi (LD) and the abdominal transversus muscle (ABTM) and the abdominal oblique muscles (ABOM) join at a raphe (R). The sheath then proceeds into three layers (deep [D], middle [M], and superficial [S]) enclosing the quadratus lumborum (QL) and the erector spinae muscles (ESM). When the muscles contract (arrows) the fascia undergoes tension and creates internal pressure within the compartment (small arrows). SC = spinal canal.



This synchrony is termed the *lumbar pelvic rhythm* (see Fig. 1-45) and is a neuromuscular activity that is “engrammed”<sup>96</sup> in the premotor brain centers with sensory feedback from the spindle system (see Figs. 1-35 and 1-36).

The engram neurophysiological concept is accepted by neurophysiologists. The engram, a neurologic aspect of memory, is first stored as a dynamic engram<sup>96</sup> within the cerebral cortex. The premotor (primary motor cortex) has recently been mapped within various regions of the cortex for hand activities.<sup>97</sup> A similar engram for the low back undoubtedly exists but remains to be confirmed.

There is a reverberating excitation from feedback circuits that bring about structural changes in the involved synapses, forming a structural engram. The genetic (memory) code has been postulated to be DNA that undergoes changes within neurones and glial cells. How learning affects these circuits remains obscure, but frequent repetition ensures fortifying the engram. An engram can be ignored, moderated, or altered.

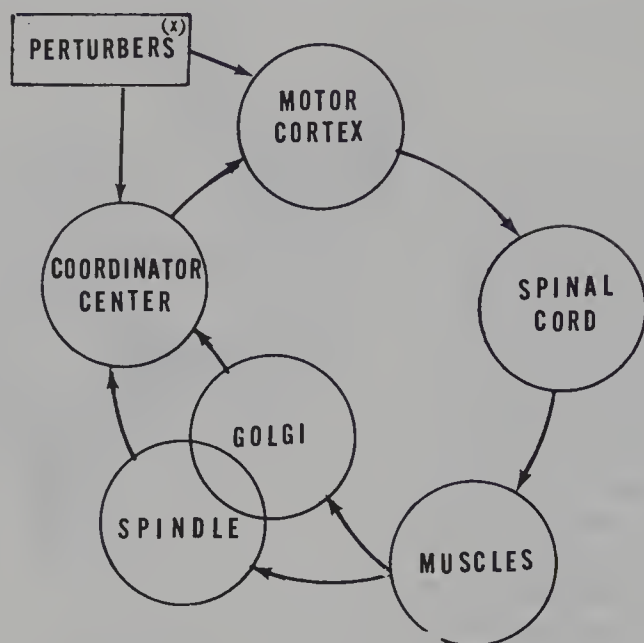


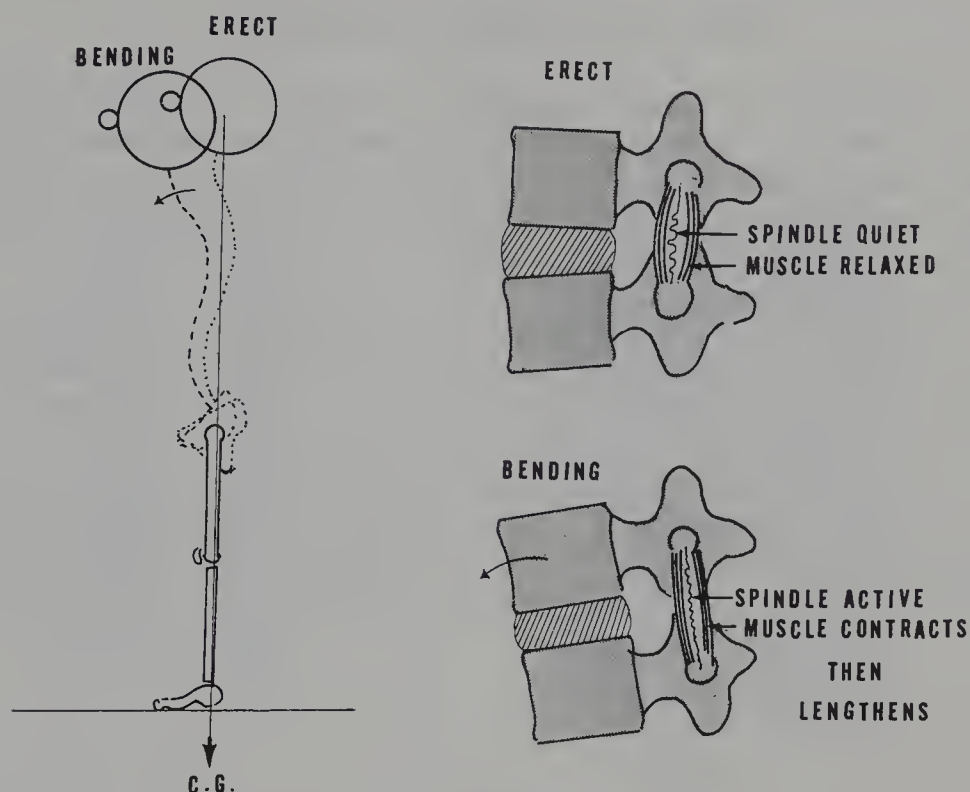
Figure 1-59. Perturber influence on neuromuscular mechanisms. The normal neuromuscular sequence of activity originating at the motor cortex transmitted to the muscle via the spinal cord, coordinated from feedback by the Golgi and spindle systems can be impaired by perturbors. Some of the perturbors are anxiety, fatigue, anger, distraction, depression, and so on.

The concept of an engram pertains to lumbar pelvic rhythm that occurs physiologically when implemented yet can be altered by perturbors (Fig. 1-59).

## LUMBAR PELVIC RHYTHM

How the low back functions is determined by basic neuromuscular activity termed lumbar pelvic rhythm.<sup>2</sup> In the erect stance, the trunk muscles are basically acting only to produce tone, and the total spine is consistent with the center of gravity. The weight-bearing support is that of intrinsic intradiskal pressure and ligamentous tone.

As a person bends forward (flexes), the erector mechanism becomes activated (Fig. 1-60) by the dormant spindle system being elongated (see Fig. 1-35). The trunk flexes as the pelvis initially remains fixed from isometric contraction of the pelvic muscles: the glutei and hamstrings. The trunk flexes as the extensor muscles decelerate the flexion.



**Figure 1-60.** Initiation of forward flexion. In the erect stance over the center of gravity (CG) the erector spinae muscles are relaxed and the spindle system is inactive. As soon as there is the intention of flexing forward the head and upper body goes anterior to the center of gravity, initiating an immediate and appropriate activation of the extrafusal muscle fibers by initiating spindle activity. The extrafusal fibers elongate as the total body flexes. (From Cailliet, R: *Soft Tissue Pain and Disability*, ed 2, FA Davis, Philadelphia, 1988, with permission.)

This deceleration is a neuromuscular mechanism that is engrammed in the central nervous system and can be affected by perturbers and conditioning of the involved tissues. The ligaments and fascia that were also relaxed become activated as they passively elongate. Once the spine is fully flexed, the muscle becomes quiet and the fascia and ligaments become fully elongated (see Fig. 1-40).

The presence of perturbers, faulty lumbosacral rhythm, and poorly conditioned tissues will be discussed further in the text, as will the psychological aspects of function.





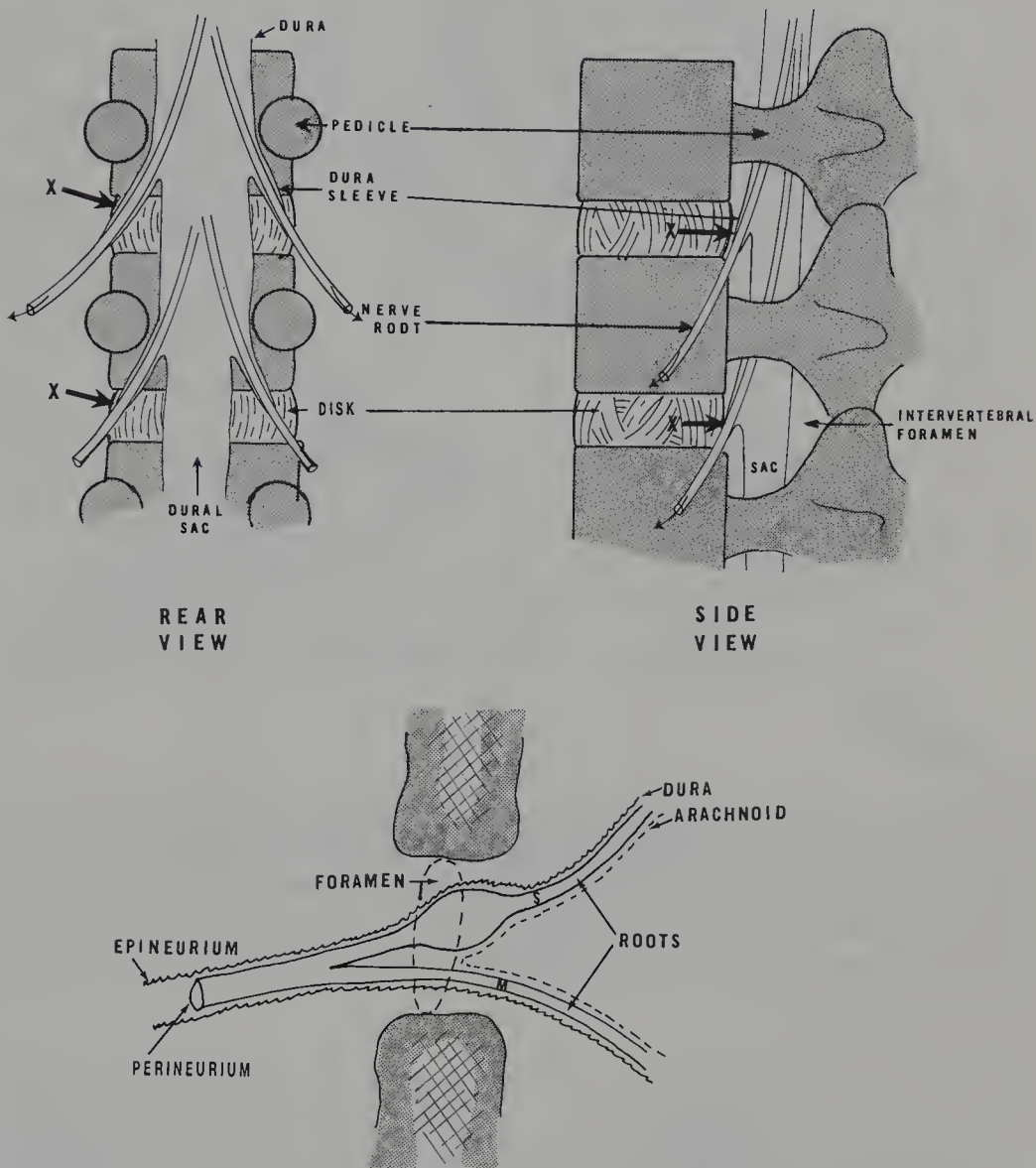


Figure 1-62. Dural and arachnoid sheaths of the nerve root. The sensory nerve roots (S) and the motor nerve roots (M) merge at the intervertebral foramen. The arachnoid terminates at the foramen, whereas the dura continues and becomes epineurium and perineurium.

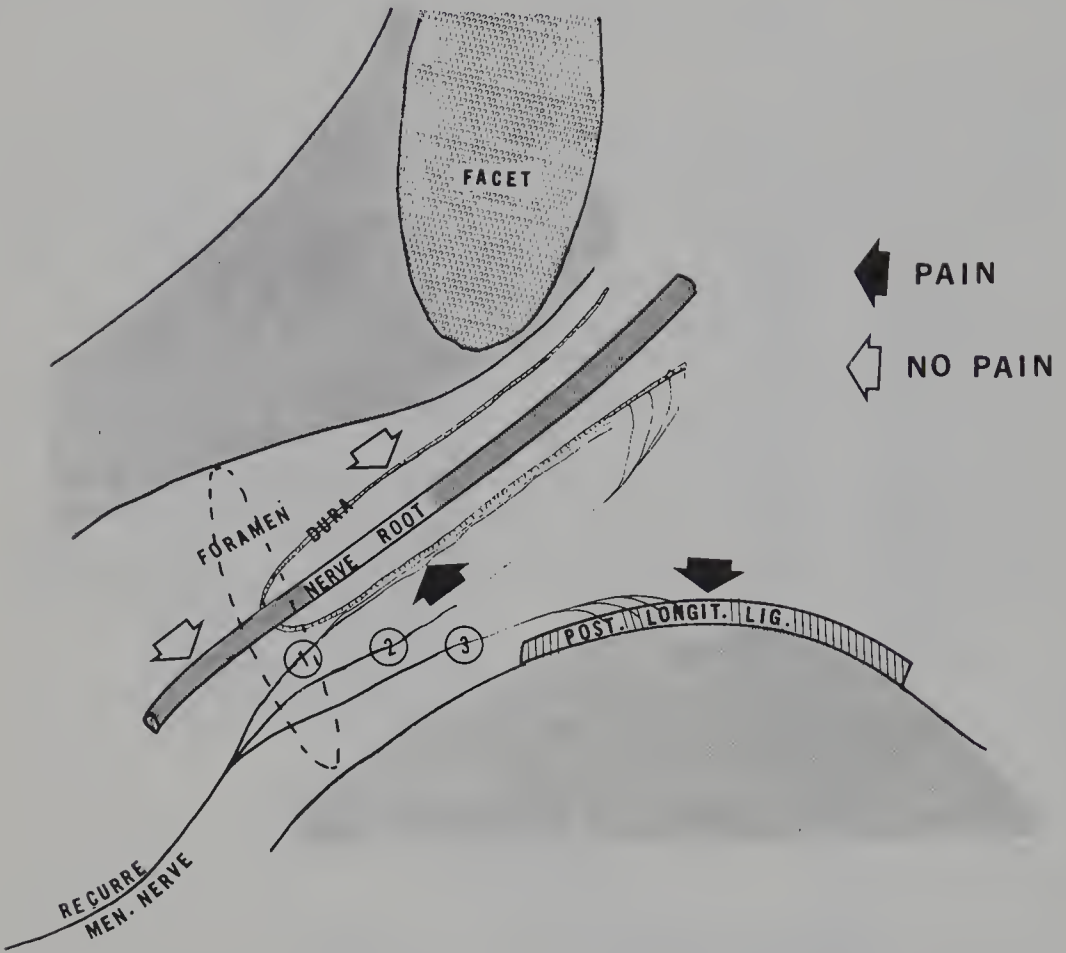


Figure 1-63. Dura accompanying nerve root. The dura accompanies the nerve root through the intervertebral foramen along with the recurrent meningeal nerve. The meningeal nerve divides into three branches: branches 1 and 2 supply the dura and branch 3 supplies the posterior longitudinal ligament. The posterior dura receives no innervation and thus is insensitive. The tissues innervated are sites of nociception.

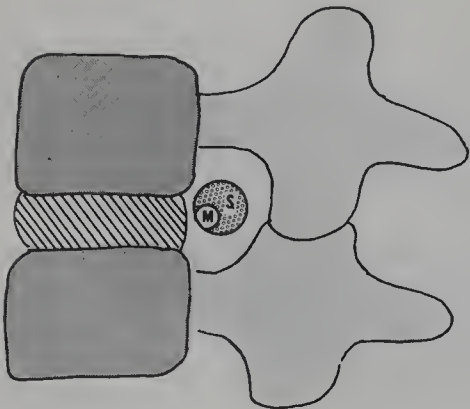
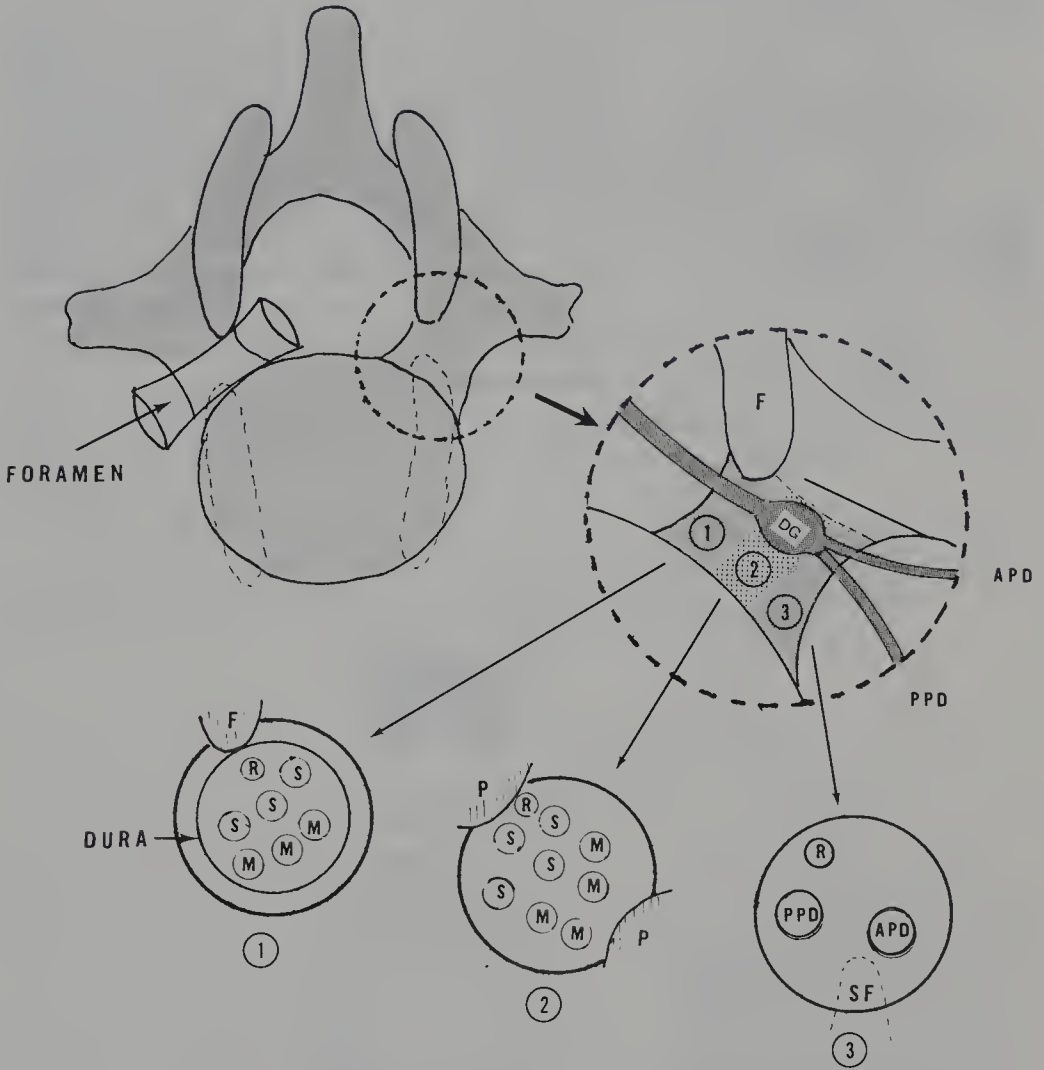


Figure 1-64. Sensory motor aspect of a nerve root. Within the intervertebral foramen the motor roots are smaller and closer to the disk whereas the sensory fibers are more numerous and within the posterior recess of the foramen.



**Figure 1-65.** Neural contents of the intervertebral foramen. The upper figure shows the foramen (*dotted circle on the right*) as a funnel shaped tubular opening (*left*). The figure within the dotted circle shows the contents with the facet (F), the superior facet (SF) of the inferior vertebra, and the pars portion (P) of the lamina. The dorsal root (DG) and the contents of the foramen (1, 2, and 3) reveal the contents of the root in those foramenal segments. The sensory roots (S), recurrent nerve of Luschka (R), motor (M), posterior primary division (PPD), and anterior primary division (APD) are also depicted.

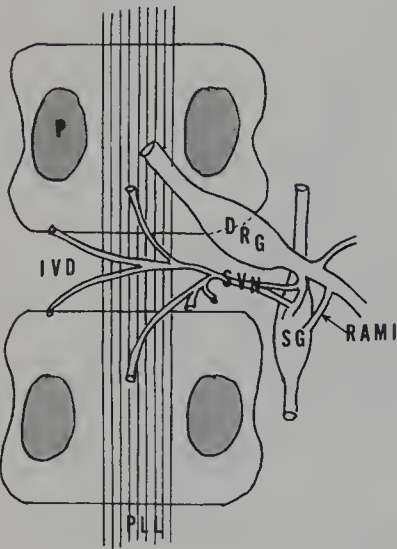


Figure 1-66. Sinuvertebral nerve, the pedicle (P), and the intervertebral disk (IVD). The dorsal root ganglion (DRG) emerges laterally giving a ramus branch to the spinal ganglion (SG) from which emerges the sinuvertebral nerve (SVN). This sinuvertebral nerve, originating from the white ramus, innervates the dura, the posterior longitudinal ligament (PLL), and conceivably the very outer layers of the annulus.

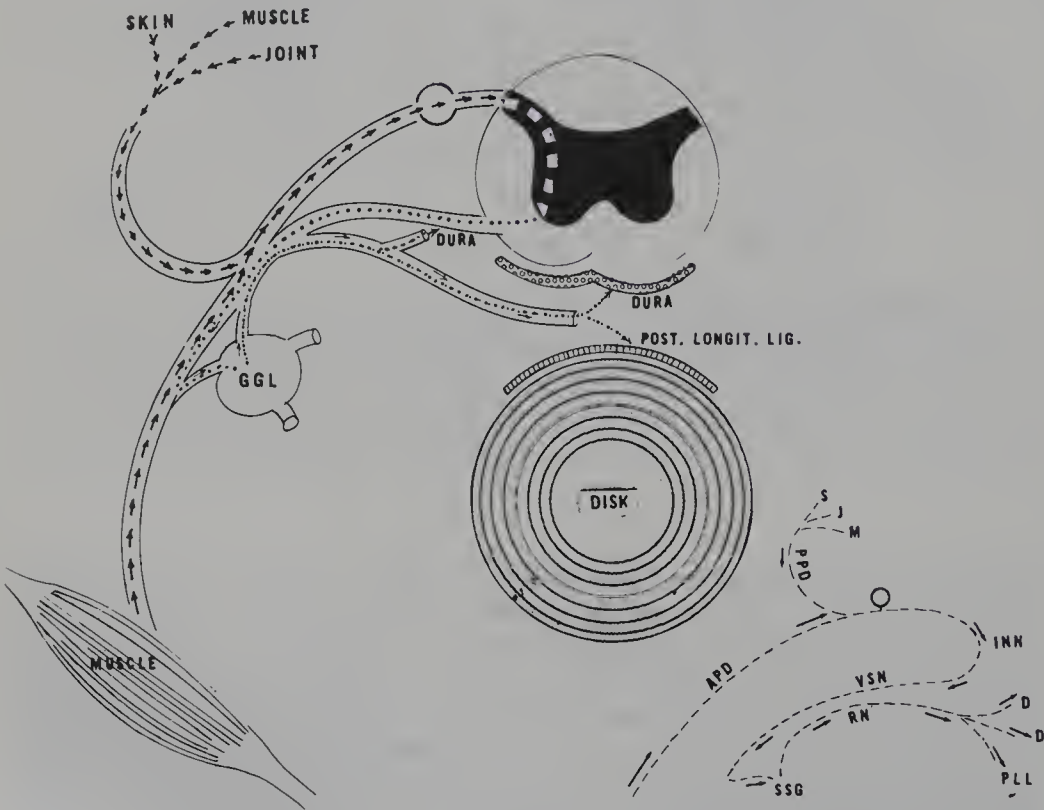


Figure 1-67. Innervation of the recurrent nerve of Luschka. (APD = anterior primary division; D = dura; GGL = sympathetic ganglion; INN = internuncial neuron; PPD = posterior primary division; PPL = posterior longitudinal ligament; RN = recurrent nerve of Luschka; SSG = sensory sympathetic ganglion; VSN = ventral sensory nerve.)

fibers but not to the center annular fibers or the nucleus. There are encapsulated nerve fiber endings within the outer annular fibers that are considered to subserve proprioception. The outer annular fibers of the intervertebral disk were initially implicated when Steindler and Luck resolved low back pain and sciatica by injecting a local anesthetic into the disk.<sup>98</sup> Direct pressure on the disk also caused low back pain. Motor fibers functionally separated.

During surgery for a pathological low back with sciatic radiculopathy, the nerve is either compressed, deformed, or stretched by the protruded disk but the nerve root is also congested and swollen if the symptoms have been pain. The nerve viewed microscopically shows demyelination and degeneration. Excessive venous entry and dilatation is also seen.<sup>99-102</sup>

There appears to be a direct pathway between the nerve root and the nucleus pulposus.<sup>103</sup> In the performance of a discogram there is often a leakage of the dye into the epidural space. When injured, the disk “secretes” inflammatory material such as phospholipase A2 and cytokines.

The root level depicts the specific nerve root emerging from a specific foramen (Fig. 1-68) and its distribution is to a specific superficial area or to muscles in the lower extremity (Fig. 1-69).

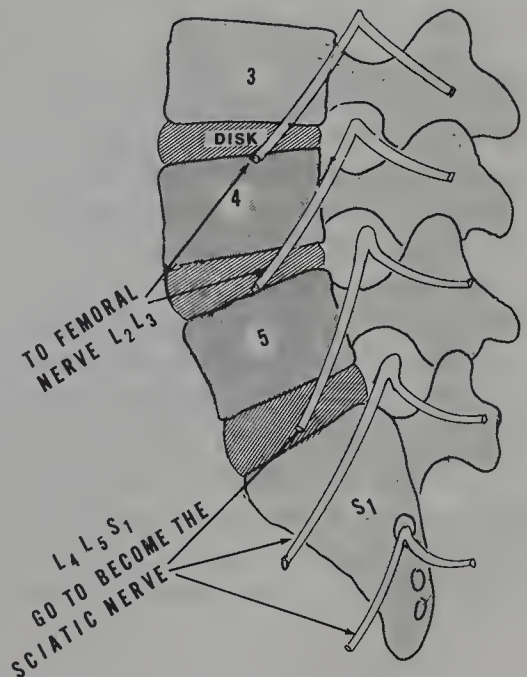


Figure 1-68. Sciatic nerve roots. The fourth and fifth lumbar and the first sacral roots emerge from their specific foramina and descend to form the sciatic nerve. They innervate the sensory and motor aspects of the lower leg, foot, and toes. The second and third lumbar nerve roots form the femoral nerve and descend the anterior thigh to innervate the upper portion of the lower extremity.



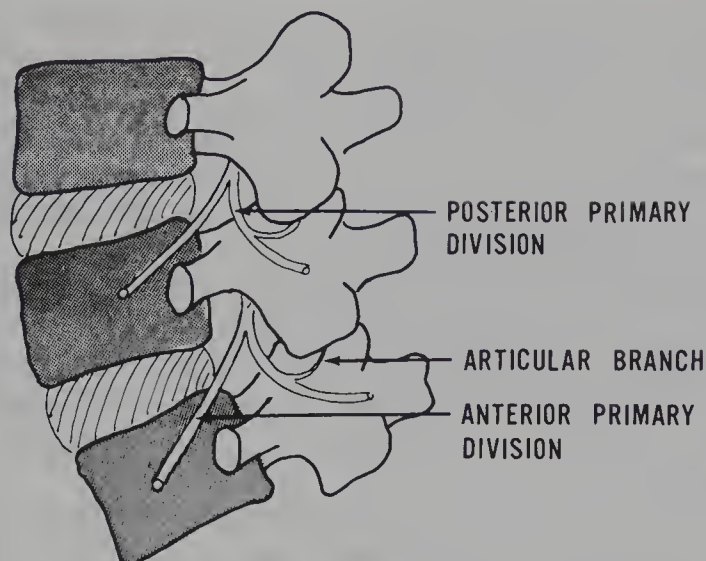


Figure 1-69. Division of nerve roots. When emerged from the foramen, the nerve roots divide into the anterior primary division and posterior primary division. A small articular branch is sensory to the facets.

The nerve roots are somatic and sympathetic in their structure and function. The lumbar sympathetic chains descend along the anterolateral aspects of the lumbar spine. (Fig. 1-70). They typically join the lumbar ventral rami (Fig. 1-71) just outside the foramina (Fig. 1-72).

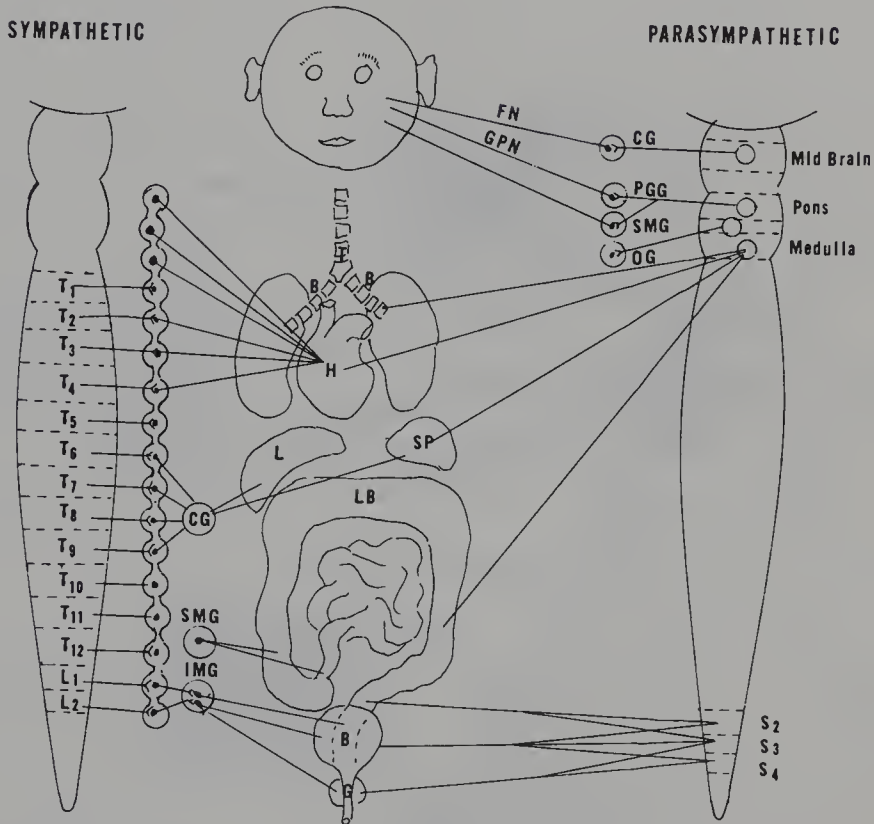
Pain is transmitted via sympathetic nerves, and chronic pain and sympathetic mediated pain have a postulated mechanism by which somatic sensation ultimately is considered pain. (Fig. 1-73).

Trauma to peripheral tissues (i.e., tissues within the functional units)<sup>106,107</sup> has been postulated to be chemicals (Fig. 1-74). Peripheral trauma also elicits nociceptors that activate the sympathetic nerve endings (Fig. 1-75).

Recent anatomic dissections, magnetic resonance imaging studies,<sup>108</sup> and provocative discograms have contributed to our understanding of the pathomechanics of discogenic low back pain. The other numerous tissue sites causing low back pain are being ascertained.

The relationship between pain and the nerve root<sup>109</sup> remains an enigma. Radicular pain is probably the most disabling symptom in low back pain from discogenic syndrome or degenerative spine disease. It is of interest that compression of the nerve root can exist without pain.





**Figure 1-70.** Autonomic nervous system. The autonomic nervous system (ANS) arises from the lateral horn cells of the thoracic cord (T) that emerge to synapse in the sympathetic chain, which then synapse with the celiac ganglion (CG), the superior mandibular ganglion (SMG), and the inferior celiac mesenteric ganglion (IMG). The ANS innervates the bronchi (B), heart (H), liver (L), spleen (SP), large bowel (LB), bladder (B), and rectum and sigmoid (G). The parasympathetic system arises from the midbrain, pons, medulla, and the sacral nerves (S<sub>1</sub>, S<sub>2</sub>, and S<sub>3</sub>). These innervate the face and head through the facial nerve (FN), glossopharyngeal nerve (GPN), otic ganglion (OG), celiac ganglion (CG), and posterior and superior mandibular ganglia. The sacral outflow (S<sub>2</sub>, S<sub>3</sub>, and S<sub>4</sub>) innervate the urinary tract. (From Cailliet, R: *Pain: Mechanisms and Management*, ed 1, FA Davis Philadelphia, 1993, p 30, with permission.)

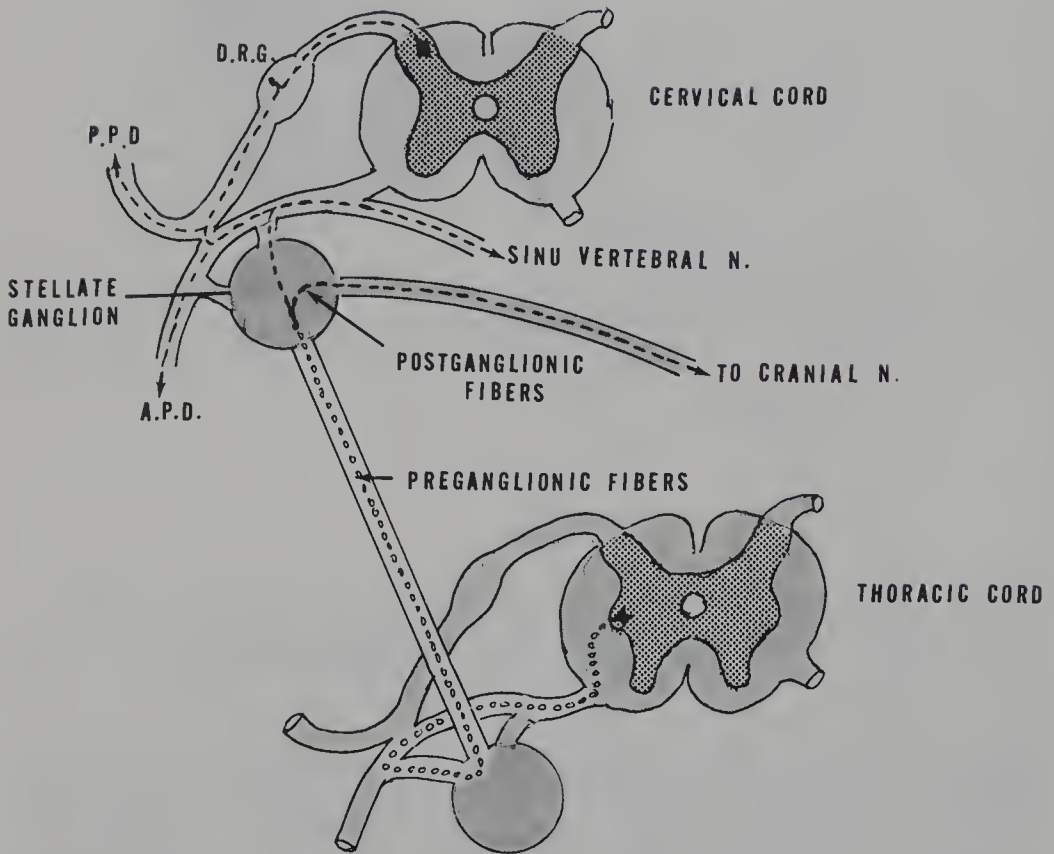


Figure 1-71. Sympathetic nervous system. The preganglionic white fibers originate from the intermediolateral horn cells of the thoracic cord and ascend to the stellate ganglion where they synapse with the postganglionic gray fibers. These autonomic nerve fibers accompany the somatic nerve when they divide into anterior primary (APD) and posterior primary (PPD) divisions. The afferent fibers enter the dorsal horn of the cord through the dorsal root ganglion (DRG).

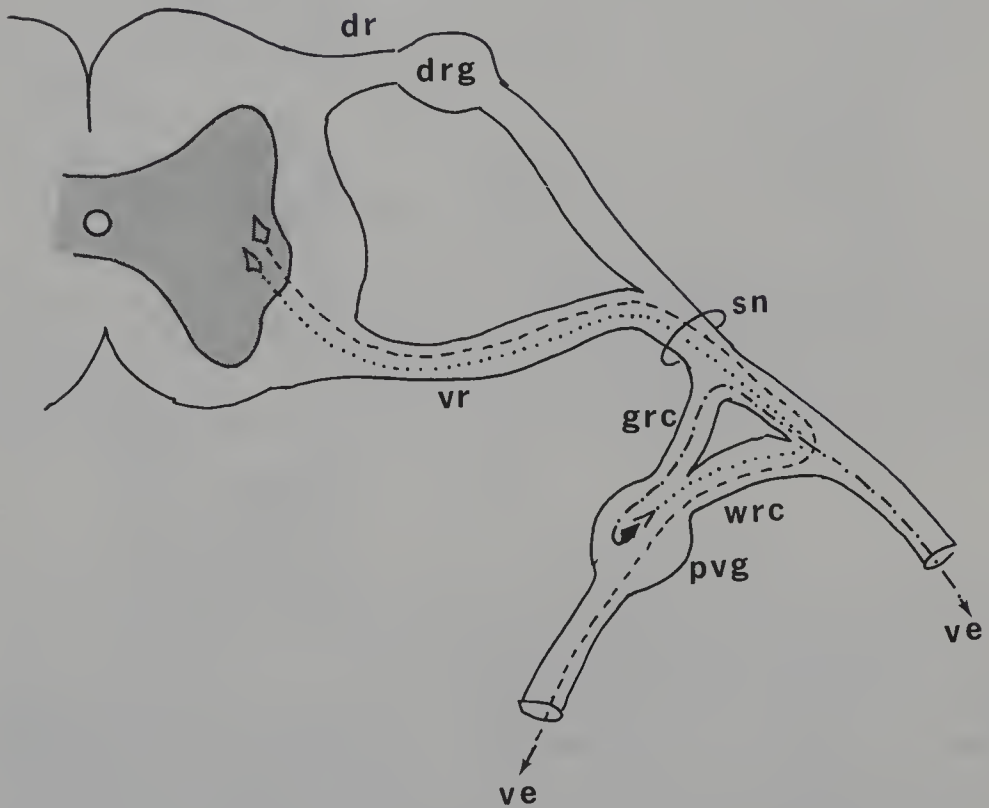


Figure 1-72. Autonomic fibers in a spinal nerve. From the lateral horn cells in the cord gray matter, autonomic fibers descend the ventral root (VR). The *dotted line* shows the fibers entering the white ramus communicans (WRC), synapsing, and leaving the paravertebral ganglion (PVG) through the gray ramus communicans (GRC) (*dotted lines*) to become a visceral efferent nerve (VE). Some autonomic fibers (*dashed line*) leave the lateral horn cells and proceed to become a visceral efferent nerve without synapse.

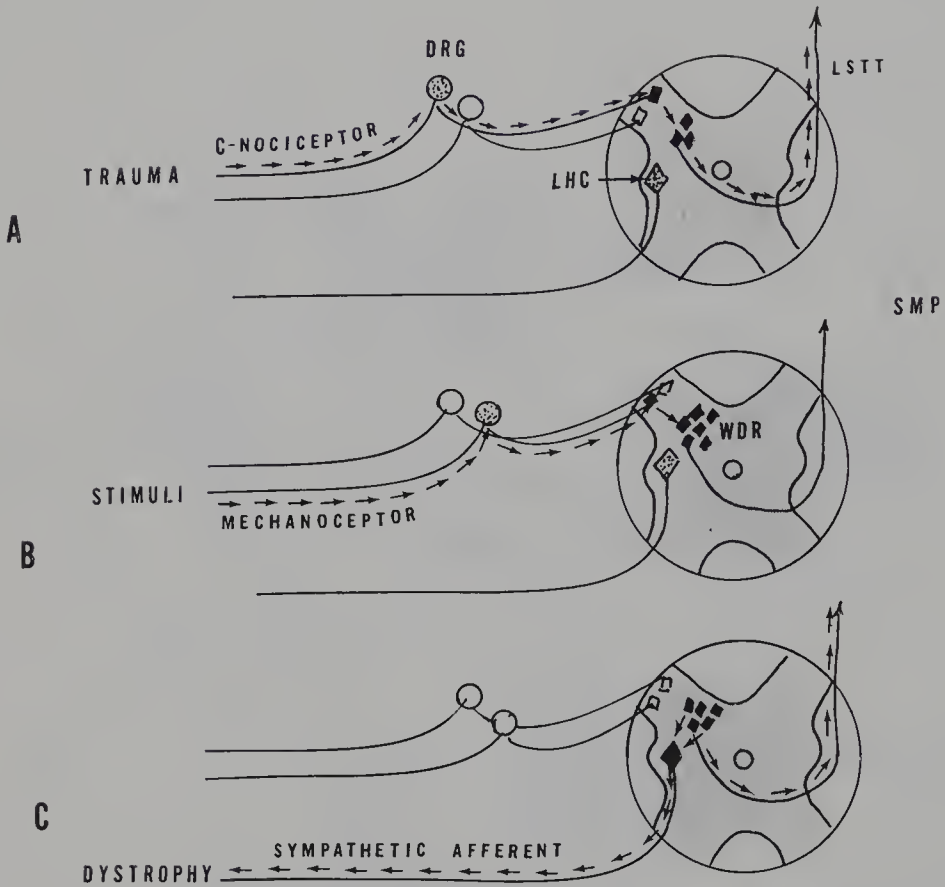


Figure 1-73. Postulated neurophysiologic mechanism of sympathetic mediated pain (SMP). The transmission via C-nociceptor fibers (A) of impulses from the peripheral tissues that have been traumatized and created peripheral nociceptor chemicals (see details in text). These impulses pass through the dorsal root ganglion (DRG) to activate the gray matter of the cord in the Rexed layers. When sensitized, they are termed *wide dynamic range* (WDR) neurons. The WDR neurons, becoming very irritated, receive impulses from the periphery via the A-mechanoreceptor fibers (B), which normally transmit sensations of touch, vibration, temperature, and so on. When the periphery is stimulated (skin touch, pressure, or joint movement), these impulses enhance and maintain the irritability of the WDR neurons. The impulses from the WDR neurons continue cephalad through the lateral spinothalamic tracts (LSTTs) to the thalamic centers with resultant continued pain. The impulses from the WDR neurons irritate the lateral horn cells (LHC), which generate sympathetic impulses that innervate the peripheral tissues, resulting in the symptoms and findings of dystrophy (C).

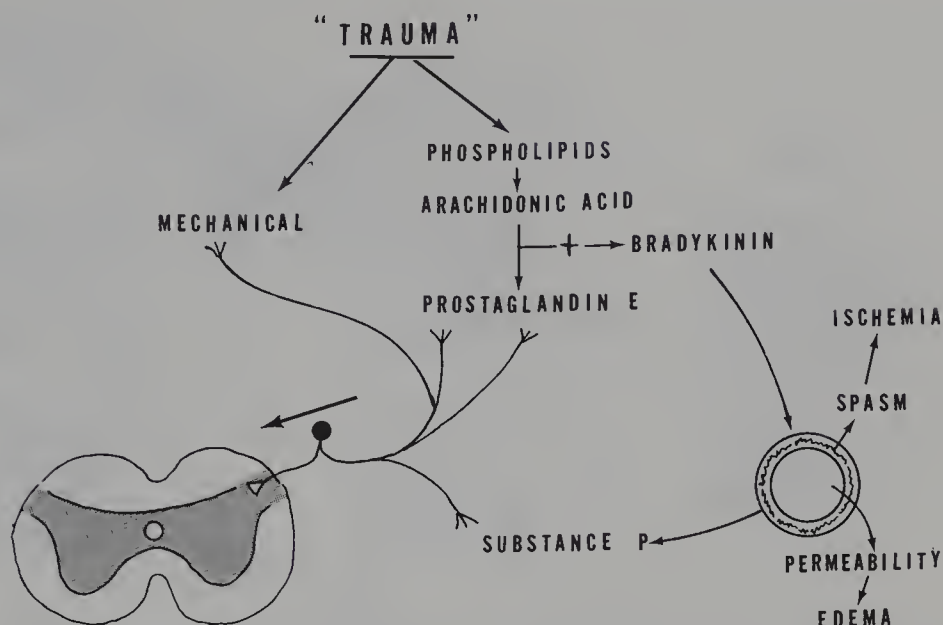


Figure 1-74. Nociceptive substances liberated from trauma. Traumatized tissue liberates breakdown products from phospholipids into arachidonic acid and ultimately forms prostaglandins. These nociceptor substances act on blood vessels causing spasm, releasing edematous fluid and release of substance P.

From 20% to 30% of patients having abnormal findings of nerve compression found on myelograms, discograms, computerized tomographic scans, and magnetic resonance images have no history of pain.<sup>110-114</sup> In humans it has been demonstrated that only an inflamed nerve root will elicit pain when mechanically irritated.<sup>115-117</sup>

The proximal part of the nerve root is enclosed within spinal fluid. The root is covered merely by a thin membrane (see Fig. 1-62) that is permeable to the cerebrospinal fluid and progresses into the peripheral nerve where it acquires epineurium and perineurium (see Fig. 1-61).

The dorsal root (DRG), which has been postulated to be a major site of radicular pain, is located within the spinal canal, the intervertebra foramen, or outside the foramen. Its position varies at various spinal levels or in various patients<sup>118</sup> but passes under the pedicle and is positioned near the disk (Fig. 1-76).

The nerve root fibers are usually in a parallel alignment, whereas a peripheral nerve is plexiform in structure.<sup>119</sup> This arrangement explains, in part, the lack of elasticity of the nerve roots. The elastic limit has been shown to be 15% of its length, and if stretched, more than 21% fail. The nerve root has a stiffness and physical strength of 10% to 20% as compared with a peripheral nerve.



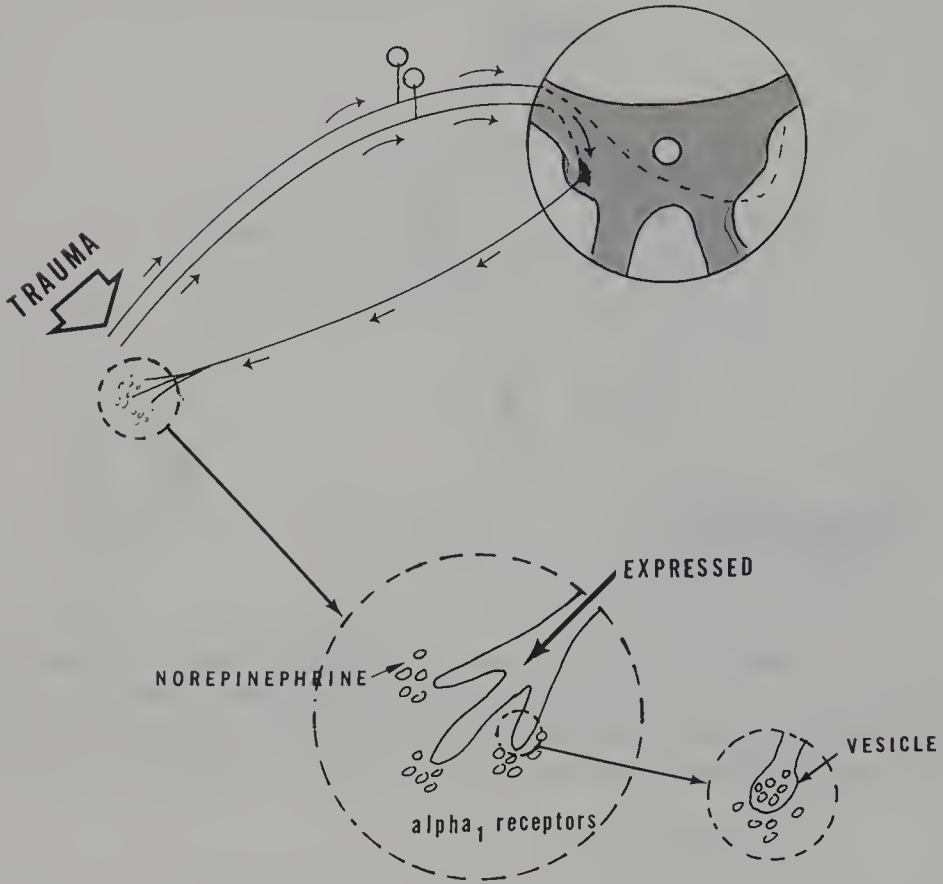


Figure 1-75. Nociceptor activity of  $\alpha_1$  adrenoreceptors. Trauma to sympathetic nerves cause formation of vesicles (*dotted circle*) that contain norepinephrine. The liberated norepinephrine reacts with  $\alpha_1$  receptors creating causalgic pain.

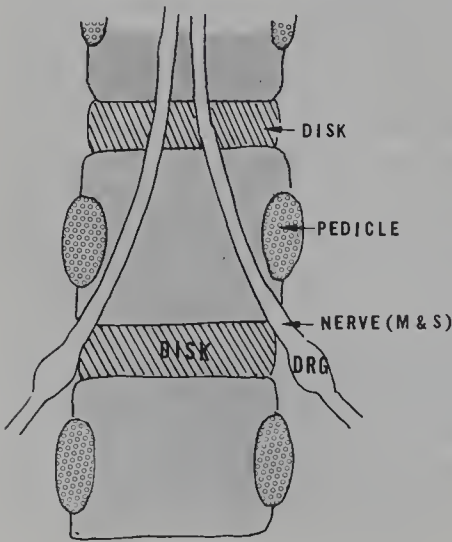


Figure 1-76. Relationship of dorsal root ganglion to the intervertebral disk. As the motor (M) and sensory (S) nerve root emerges through the foramen between two adjacent pedicles the dorsal root ganglion lays near the disk.

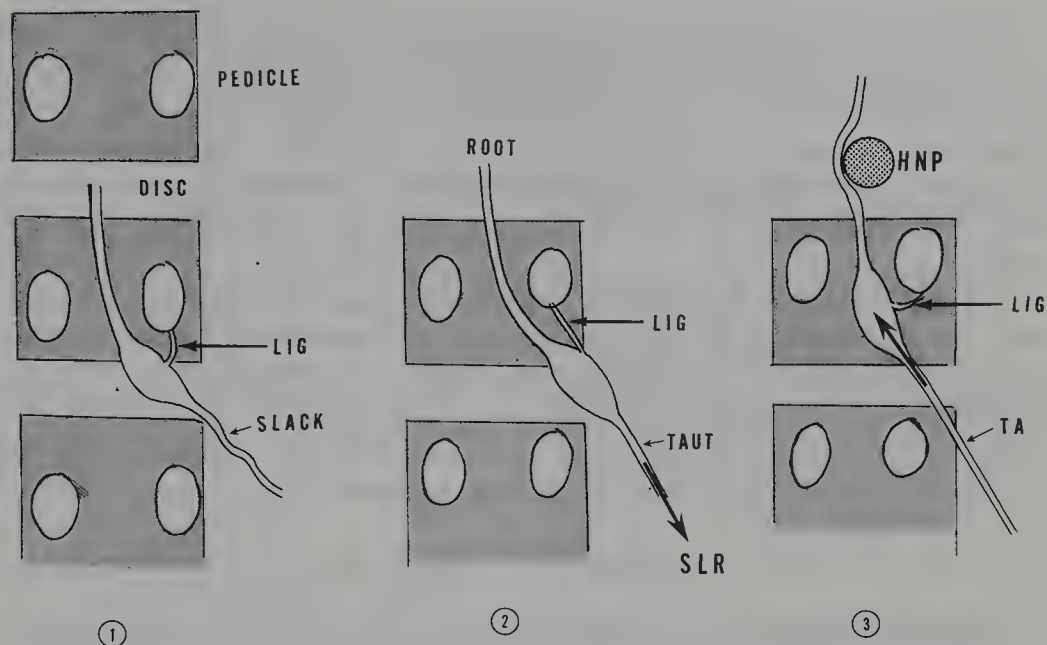


Figure 1-77. Nerve root fibrous attachments. The nerve root normally has a slack fibrous attachment at a foramen. When raising the leg (2) the nerve root migrates distally and causes the fibrous attachment to become taut, hence causing tension on the root. In the presence of a herniated nucleus pulposus (HNP) the nerve root is made more taut (3).

The nerve root dura is attached by fibrous fibrils at its orifice in the cervical spine (Fig. 1-77) and these have been identified in the lumbar region. By these attachments no significant movement of the nerve root and its dura is permitted but allows traction and compression even in its limited motion.

The dural sheath is richly innervated on its ventral surface but sparsely so in its dorsal surface.<sup>121</sup> The nerve roots contain both somatic and sympathetic fibers.

## SUMMARY

The functional anatomy of the lumbosacral spine, both static and kinetic, is the basis for evaluating the mechanisms by which the spine normally functions and malfunctions. The tissues involved are the basis for nociception when damage occurs and must be recognized in a meaningful clinical examination. The history given by the patient explains how the spine was dysfunctional, but only by knowing how the spine normally functions, can dysfunction be fully recognized.

Examination of a patient merely indicates that the impairment be recognized and understood. The disability that results from the impairment ensues from the evaluation. Structural changes must also be recognized and properly placed in the total equation toward diagnosis. Unless impaired physiology and disrupted functional anatomy is appreciated, no meaningful corrective measures considered therapeutic can be applied.

Pain leading to disability and allegedly the sequela of impairment must imply that a sensitive tissue has been involved. The neurologic involvement and its psychological consequence are the bases of understanding the disability and impairment relationship of tissue injury.

All subsequent chapters have their basis on this appreciation of functional neuro-anatomical-psychological physiology.

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## CHAPTER 2

# Psychological Aspects of Low Back Pain

After a large chapter devoted to the mechanical, structural, and neurological aspects of low back pain and leg pain emanating from the low back, it may appear strange that Chapter 2 is devoted to the psychological aspects of this disabling entity.

The costs of medical care and the outcomes assessment have become of paramount importance in general medical care. Control of medical costs involves hard choices related to availability of services, rationing of these resources, burgeoning of administrative costs, and public expectations, many of which are unrealistic.<sup>1</sup>

For centuries the psychological aspects of low back pain have been entertained, interpreted, and disputed yet remain ambiguous, poorly understood, and inadequately implemented in treating the patient with low back pain.

Pain, especially chronic pain, has become prominent in medical research and clinical application. In recent decades volumes of medical literature have been written and numerous medical specialties have become devoted exclusively to this aspect of neuromuscular functions.

As pain is becoming a medical entity there are all kinds of control mechanisms being advocated, many of which can be justified on an altruistic and humanitarian basis, but regarding low back symptoms “return to work” and its costs are prevalent in society’s decisions.

In considering acute low back pain, one must keep the nociceptive aspect in mind. At this state the purely sensory aspect of pain can be accepted to resolve the pain and its resultant disability. Chronic or persistent pain and disability are largely encountered when appropriate acute measures are not observed and implemented.



In newer findings on the neuroanatomical basis of pain, both peripheral and central mechanisms have added to the comprehension of low back pain: acute and chronic.<sup>2</sup> The psychological aspects of low back pain also includes the mechanism of injury and the prolongation of pain into chronicity after the acute insult has passed.

The psychogenic aspects of low back pain also have become predominant in its evaluation, and all psychogenic aspects of low back function, dysfunction, and pain interpretation need to be addressed.

A possible mechanism of low back pain from psychogenic motor unit activity has been proposed in which prolonged muscle contraction from purely psychogenic influences can be a possible injury mechanism.

It has been accepted for a long time that psychogenic muscle tension exists in sedentary work tasks that otherwise have low postural or physical demands.<sup>3,4</sup> There are also postural positions that are aggravated by tension. Muscular activity is typically only 5% of the energy level of voluntary activity.

Psychogenic muscle tension has been associated with musculoskeletal pain syndromes.<sup>5,6</sup> The proposed mechanism is repeated, prolonged activity in a small pool of low-threshold motor units, which gradually overloads the affected muscle fibers. This phenomenon has been postulated in occupational activities of the neck and shoulder.<sup>7-10</sup>

The mechanism of this psychogenic muscle activity causing neuromuscular symptomatology is prolonged activity in a few motor units rather than a shift in activity of different units within a large pool.<sup>11</sup> The activity in these small motor units involves type I motor fibers that undergo "overload".<sup>12-14</sup> Prolonged activity leads to muscle ischemia,<sup>15</sup> which becomes a site of nociception, pain, and further muscular activity.

This psychogenic muscular overactivity has been documented in neck and shoulder muscles but a similar occurrence may well exist in the erector spinae muscles and needs exploration.

Besides the acceptance of psychogenic muscle tension in the causation (mechanism) of low back dysfunction with resultant pain, the interpretation of low back pain demands a psychological assessment of the patient.

Low back pain has been depicted as a *neuro-musculo-skeletal-psychological entity* with all that this term implies. Recently the psychological component has been emphasized.

The basic disability formula of  $\text{disability} = A + B + C$ <sup>16</sup> has been advanced because so many factors influence the ultimate disability. *A* is the disease involving the physical components or essentially the impairment. *B* is the patient's psychological state: at the time of evaluation or at the time of onset. And *C* is the situation associated with the onset of pain: essentially the circumstance leading to or influencing the injury.



Each of these components are factors that need to be evaluated in determining the cause, the extent, and the significance that influence the management and the prognosis.

Disability also differs from impairment, yet too often in current clinical practice the two are equally equated. Resolution of the impairment may not significantly influence the disability, nor is the disability equal in all aspects to the impairment. Too often disability occurs without impairment, and conversely impairment often occurs without disability. The medical model today addresses disability without a careful evaluation of the impairment and vice versa; both the patient and society loses.

Psychological testing to ensure a comprehensive pain diagnosis has been given impetus by literature stating that psychological testing before surgery is valuable in assessing the ultimate outcome.<sup>17-20</sup> These reports assume that the surgery was indicated, that the patient was otherwise prepared, and that the postoperative follow-up was appropriate.

Psychological evaluation of patients with low back pain has become an integral part of evaluating, diagnosing, and formulating a treatment plan for the afflicted patient. Pain is not merely a descriptive term of the experienced nociception but is a multidimensional phenomenon. It comprises the physiological but also emotional, social, cultural, and even educational factors.<sup>21</sup>

Depression causing, enhancing, and/or following a low back injury has become a predominant factor to be considered and confronted.<sup>22</sup> The presence of depression and anxiety prolongs pain and dysfunction whether or not there has been a psychosocial problem related to the supposed incidence and whether or not there is or has been an impairment considered as nociceptive.

Psychodiagnostic tests have been well established in patients considered to have a psychiatric diagnosis but are not applicable or documented in nonpsychiatric patients, including patients who have low back pain. The current concept that patients with benign low back pain and nonconfirmatory tests are emotional problems begs the truth of all aspects of that statement.

Psychological evaluation, often undertaken late in the diagnostic and therapeutic process, has given the patient the impression that all symptoms are fabricated, "psychological," and even fraudulent instead of being used constructively to assess and treat the patient appropriately.

Many health professionals have inadequate training or experience in interpreting most psychometric instruments,<sup>23</sup> and many do not know when and why to order them. The problem is compounded by their explanation of the need and reason for the instrument and ulti-

mately their interpretation of it being discussed and explained to the patient.

Most psychometric tests are time consuming and cumbersome and not specifically directed to a patient experiencing pain from a low back incidence. A recent test has been proposed that takes some 15 to 20 minutes and is a pencil-and-paper test.<sup>24</sup> This test allegedly assesses the degree of psychological distress and how the pain influences the patient's functioning. In such a test, function as well as the meaning of pain is highlighted.

In the clinical evaluation of the patient with low back pain, the mechanisms by which the patient developed low back pain comprise the history. An appropriate examination reveals which tissues are the nociceptive tissue sites. The history and physical examination become the basis for making a meaningful diagnosis and for appropriate treatment.

Functional loss is paramount, with pain being secondary, albeit primary in the patient's mind. The pain must be addressed and thoroughly evaluated and ultimately treated. Many of the factors of history and examination, however, must be revised to alter the current method of examination, which has compounded the problem and led to inadequate treatment.

Work distress and dissatisfaction have proved to be a major component in the cause of low back pain and must be recognized and addressed in rehabilitating a physically disabled worker.<sup>25</sup> A work distress evaluation has been proposed (generalized work distress scale, GWDS), which currently can be considered a research project but has applicability in clinical evaluation in rehabilitation efforts.<sup>26</sup>

This is a simple questionnaire, with answers ranging from 1 (rarely) to 5 (most of the time), with gradient answers of severity in between. The following questions are examples:

1. I feel powerless to do anything about my work life.
2. I feel blue at work.
3. I am restless and cannot keep still at work.
4. It is easy for me to relax at work.
5. I do not sleep well at night because of work.
6. When things get tough, I feel there is always someone at work I can turn to.
7. I feel that the future at work looks bright for me.
8. I feel that I am needed at work.
9. I feel that I am appreciated by others at work.
10. I get easily upset at work.
11. I feel that I don't have a good time at work.
12. I feel that people at work really care about me.
13. I feel that my situation at work is hopeless.

There are other questions that are included in the GWDS and many that can be added or deleted. In a meaningful examination of the patient with low back injury, some of these questions need to be asked before a formal questionnaire is presented.

The psychological aspects of pain and disability must be considered in their initial manifestation. This statement may evoke a mixed reaction, inferring that pain is imaginary, that disability is entirely psychological, and that there is always a secondary gain from this pain. These are not the premises and no such thoughts are intended in discussing the psychological aspects of low back pain.

How the patient injured his or her low back must be ascertained. The statement, so often made, "I was injured" is rarely accurate. The patient is usually responsible for having incurred the injury, but this statement must not be construed as an accusation but merely as an etiologic factor.

Many if not most low back injuries occur when there is an inadvertent improper activity that occurs when the patient is tired, upset,

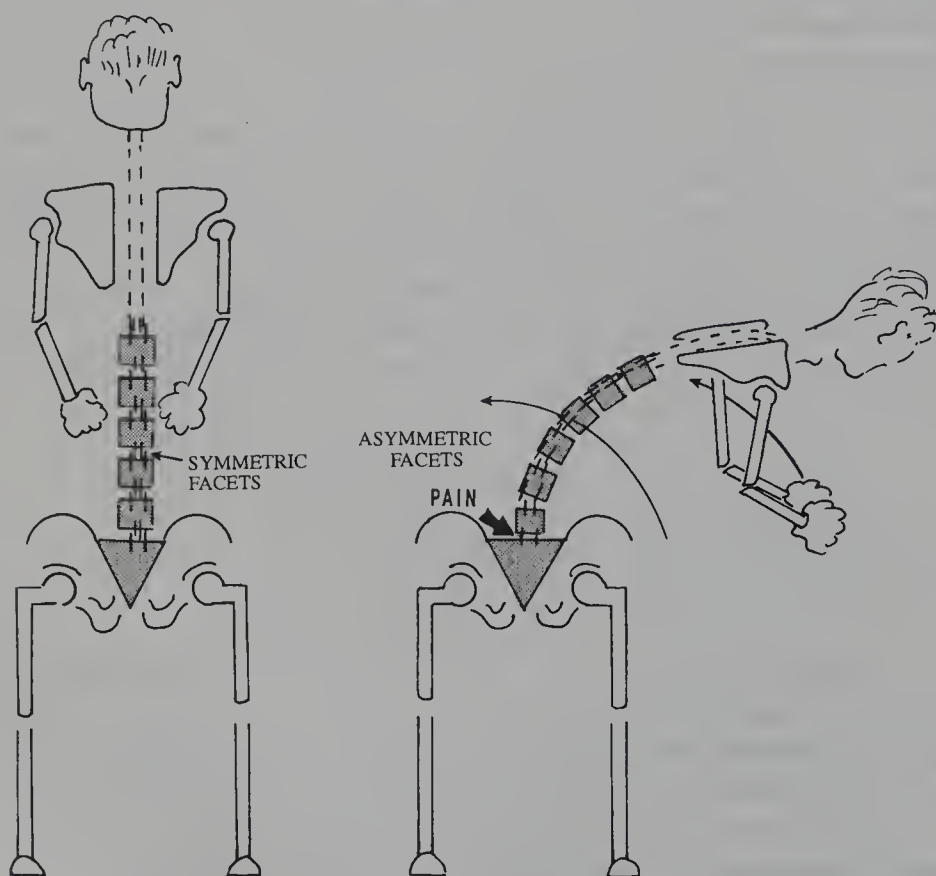


Figure 2-1. Facet alignment in flexion and rotation. This illustration depicts how the facets direct lumbosacral motion. When distracted, angry, anxious, or fatigued the person bends forward and to one side and returns to the erect posture in an inappropriate extension-derotation manner. The facets impinge with resultant pain and impairment.

distracted, angry, preoccupied, depressed, anxious, or in some way diverted. These are psychological yet manifest themselves mechanically in that they impair the normal neuromuscular functions of the low back.

A carefully elicited history makes the diagnosis of mechanism, and the examination reveals the resultant tissue impairment. The disability results from the interpretation by the patient of the severity and significance of the pain and the resultant impairment.

These neuromuscular functions have been thoroughly discussed, implying the need for good flexibility and reasonable muscle strength but more important one should follow the precise accurate movements of all the involved tissues when performing bending, lifting, reextending, prolonged sitting posture, and so on.

Examples of these psychological mechanisms resulting in low back pain are given in the following illustrations and their legends: Fig. 2-1 through 2-10.

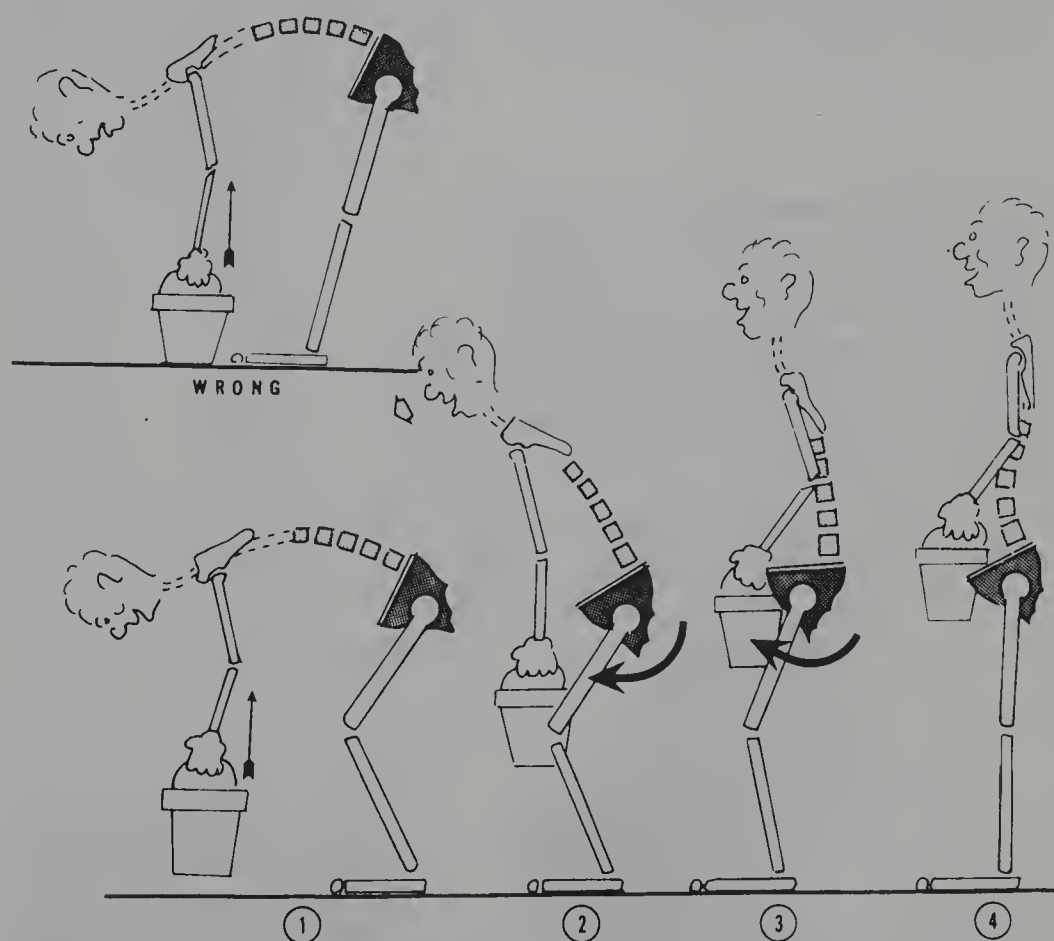


Figure 2-2. Proper lifting with flexed knees. The upper figure shows lifting from the floor with knees straight causing excessive strain on the low back. In the bottom figure the person bends the knees and slowly returns to the erect posture (1) by derotating the pelvis first (2) then reextending the low back (3) to the full erect posture (4).



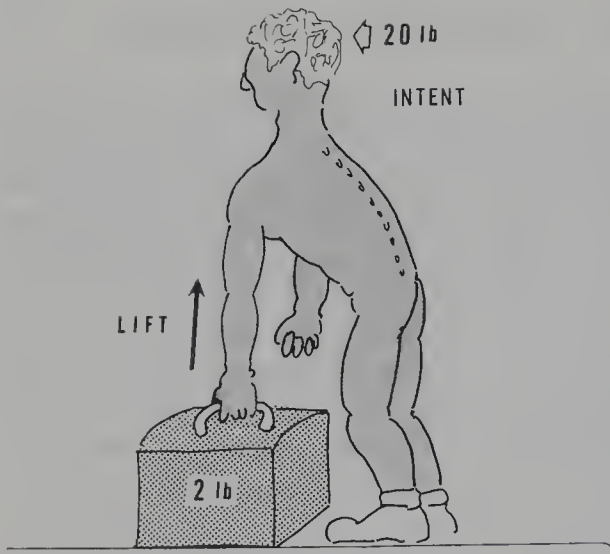


Figure 2-3. Proper judgment of effort to task. In any physical effort the mind must be preset to the task to determine the weight, the distance, and the speed of the effort. To inadequately preset the task, erratic motion or inappropriate effort is expended and tissues can be damaged.

Figure 2-4. Unexpected sneeze. An unexpected sneeze can cause a significant neuromuscular strain on the unprepared lumbosacral spine. This can also occur from any unexpected neuromuscular activity such as a slip, a fall, an external force, and so on. The unprepared body reacts with tissue reaction of inflammation.

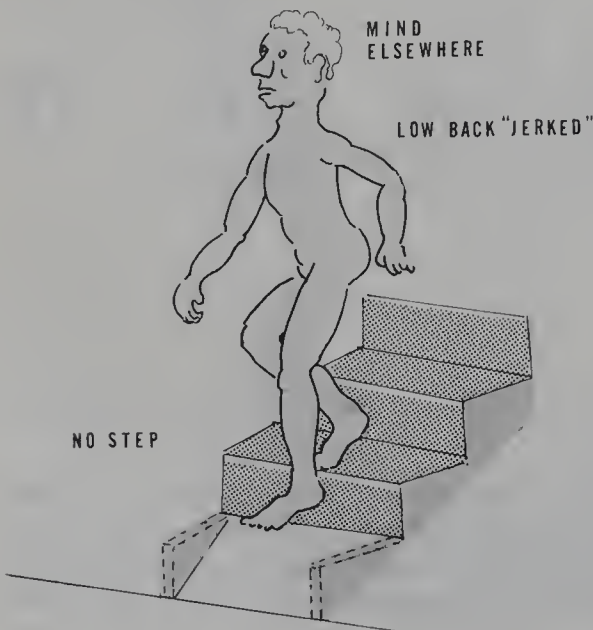
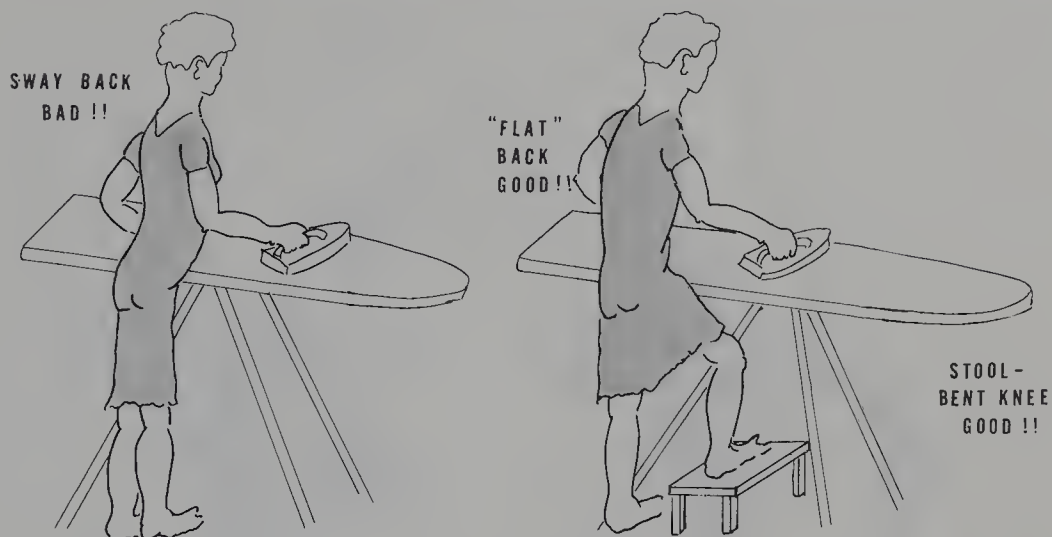
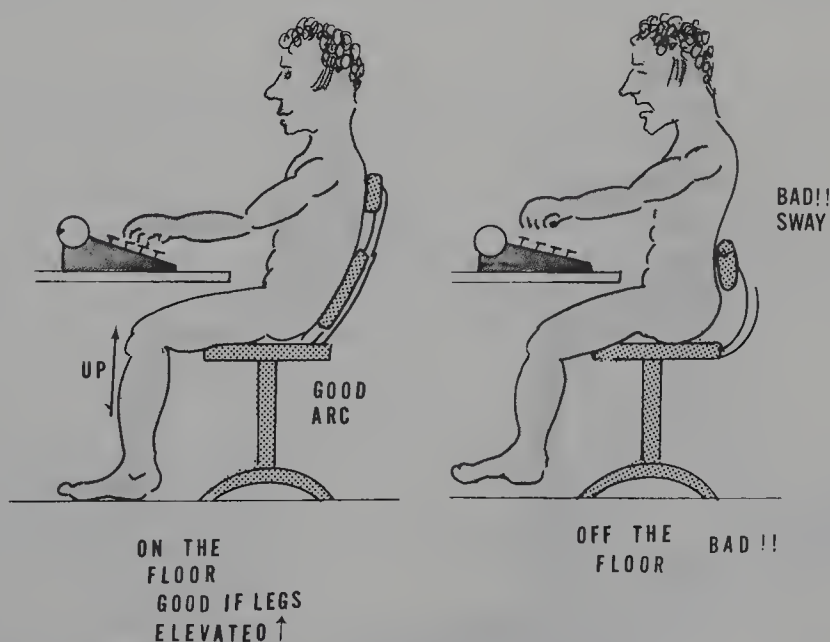


Figure 2-5. Unexpected ergonomic obstacle. In making a physical activity with an unexpected structural defect, such as a step not being there or a change in the walking surface, the neuromusculature of the low back is unprepared.





**Figure 2-6.** Faulty posture in normal activities. Faulty posture in normal activities of daily living can place a neuromuscular strain on the low back. This is true, for instance, in prolonged ironing with the board too low, too high, the legs held straight, and so on.



**Figure 2-7.** Faulty prolonged sitting posture. Sitting for prolonged periods of time with faulty sitting posture can cause low back pain. The type of chair, the height of the seat, the distance from the equipment being used, and the distance of the feet from the floor all may be conducive to the development of low back pain.

**WRONG**

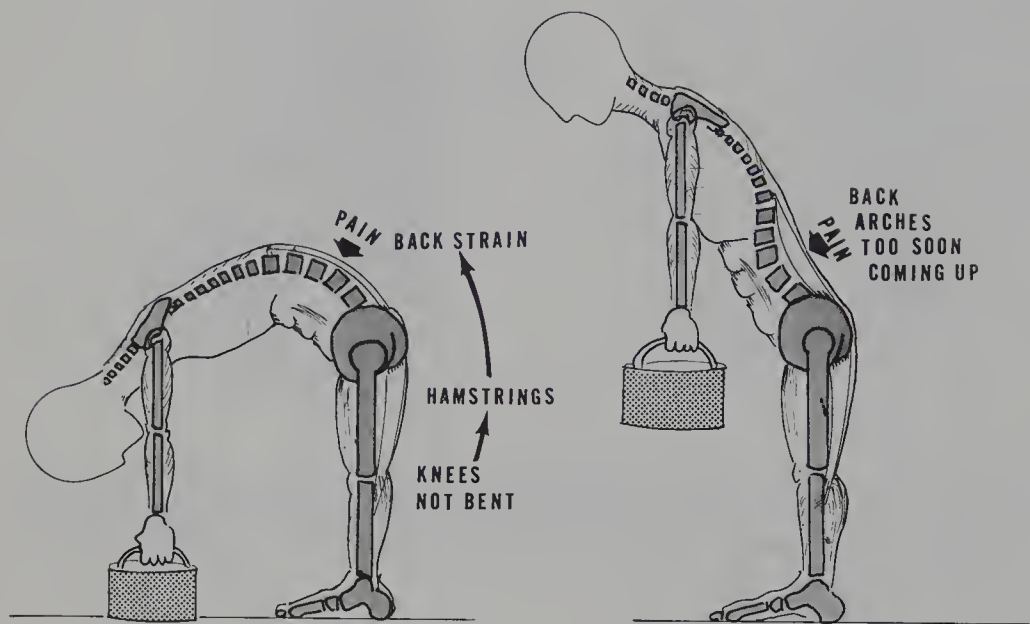


Figure 2-8. Faulty lifting technique. The left figure shows improper lifting of an object far from the center of gravity with the legs not bent and lifting exclusively with the low back. The right illustration shows the low back being reextended prematurely in the lift effort, causing the erector muscles to do most of the extension in the lift. This is a violation of the lumbar-pelvic rhythm.

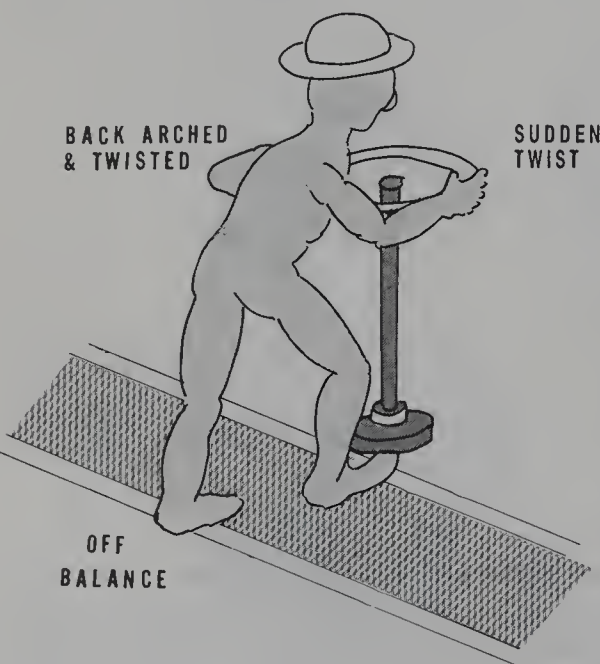
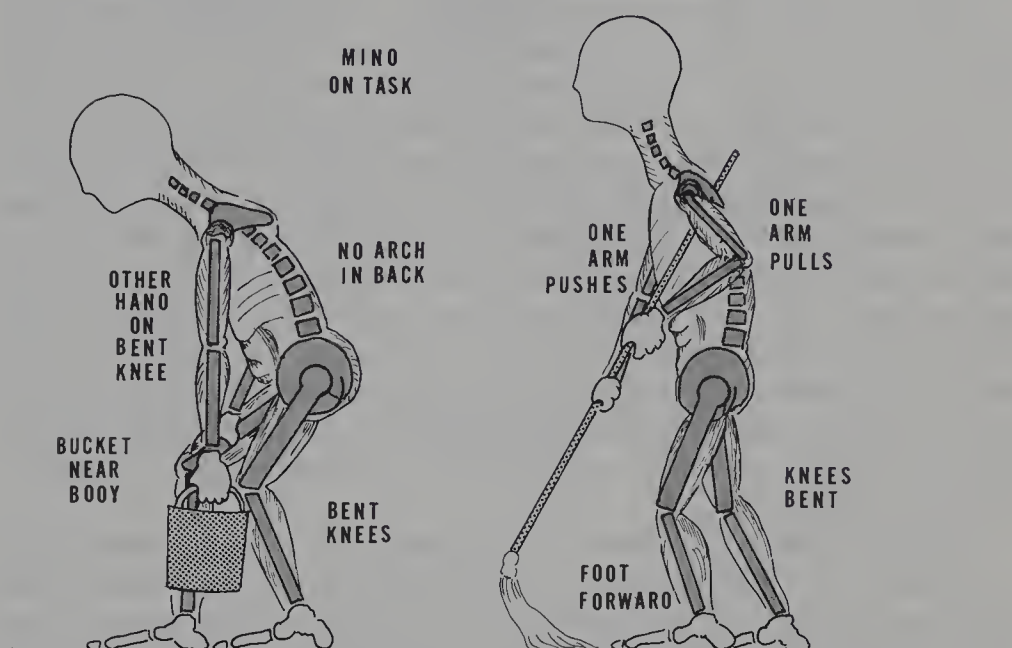


Figure 2-9. Faulty occupational activity. The low back can be injured in an occupational situation where the standing room is too narrow, the floor surface slippery, the object being manipulated too far from the center of gravity, and so on.

**GOOD**

**Figure 2-10.** Activities of daily living done properly. Activities such as lifting a small object from the floor, sweeping, or mopping must be done properly and always with the mind on the activity.

Proper functional activity training should be included in appropriate treatment protocols (see Chapter 10). Teaching enforces the neurophysiological engram existing in the cortex (see Chapter 1), and avoidance of perturbers is essential. The event elicited in a proper history and related to the resultant physical examination findings aids in the diagnosis and in explaining the cause of the low back pain to the patient.

This history informs the examiner but should be clearly discussed with the patient as to why and how the event occurred. This information should be instructional and not accusatory and relate to prevention of recurrence and avoidance of existing factors that may be remedied. By discussing how the frame of mind at the time of injury influenced the ultimate pain and disability, one can develop a therapeutic approach to the psychological aspects of the problem.

## ACUTE PHASE

Assuming that there is undoubtedly a nociceptive aspect of acute low back pain, these manifestations must be recognized and appropri-

ately addressed. The psychological aspects of acute pain exist, but their clinical nociceptive manifestations must be addressed simultaneously.

Pain remains the presenting complaint, and disability results from the impairment. From the very onset this pain must be addressed but always keeping in mind that improper care, attention, and discussion can initiate chronicity or excessive disability for the incurred injury.

Acute pain initiates a neurological transmission.<sup>27</sup> The locally liberated neurotransmitters (see Fig. 1-73 and Fig. 2-11), with their further transmission to the dorsal root ganglion, then the dorsal columns (Fig. 2-12) and wide dynamic range ganglia unto the limbic system are well accepted. It is also known and accepted that interruption of these pathways (Fig. 2-13) prevents continued transmission, and that further nociception transmission ultimately causes the intermediate transmission systems to become generators of pain. Therefore, early and adequate interruption of local nociception is mandatory. This will be discussed in Chapter 10.

The psychological aspects of acute pain are the interpretation of the significance, the severity, the resultant impairment and disability, the anger and anxiety over its occurrence, and the fear of recurrence. These must be addressed and understood by the patient so that inappropriate reaction does not occur, acceptance of an active treatment protocol is accepted, and chronicity is avoided.

The question of whether or not chronic pain is necessary<sup>28</sup> was expounded by the author in 1979 with the basis that inappropriate, inadequate, unnecessary, excessive treatment based on an inaccurate diagnosis resulted in many if not most cases of chronic pain. The low back etiology notwithstanding, this principle applies.

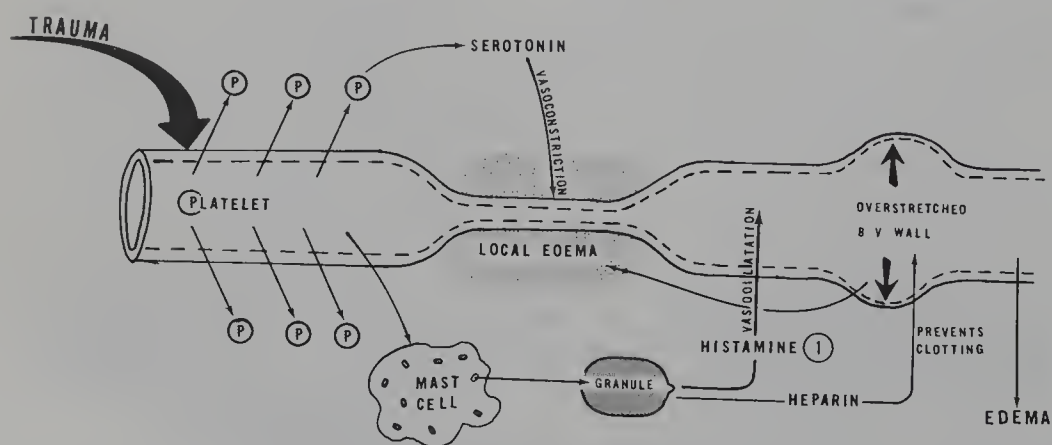


Figure 2-11. Schematic concepts of vasochemical sequelae of trauma. Trauma causes microhemorrhage or macrohemorrhage, which releases serotonin that causes vasoconstriction and also releases mast cells. The granules of these mast cells release histamine, which causes vasodilatation with resultant edema.

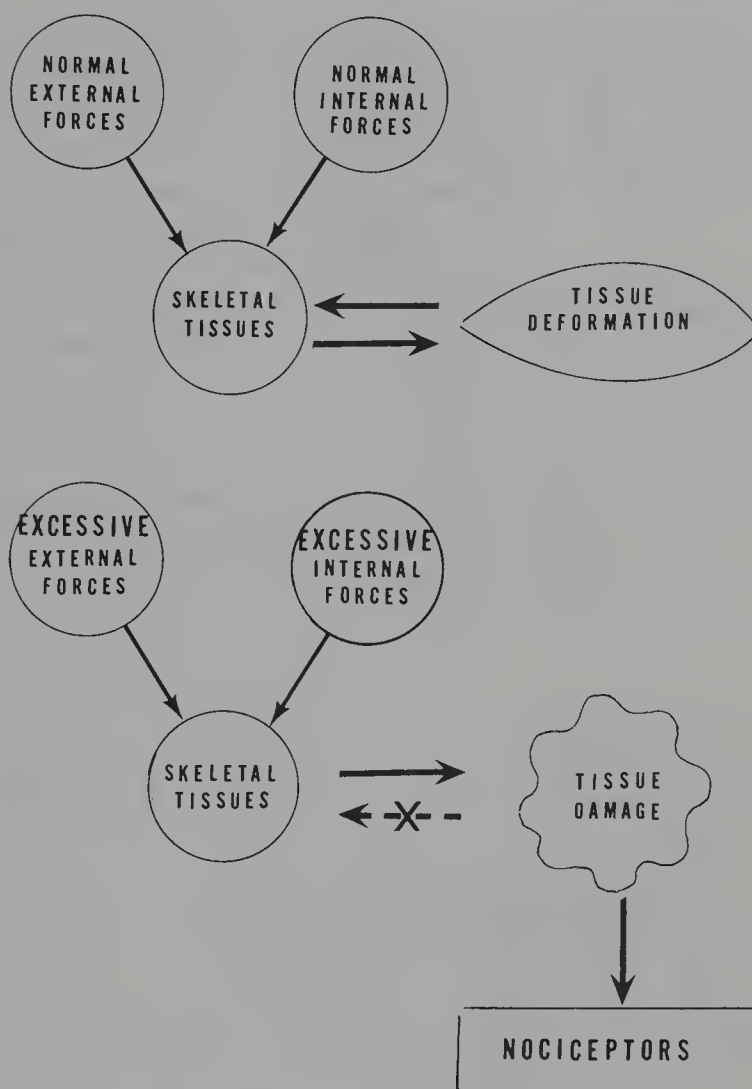


Figure 2-12. Sequence of tissue damage creating nociceptors. The upper figure shows tissue reaction to normal external and internal forces that may deform tissue with return to normal status. The lower figure depicts excessive forces on skeletal tissues that cause a certain degree of irreversible damage (*broken line*). These tissues do not return to their normal state and release nociceptors.

With the lack of objectivity of most symptoms and clinical findings, as has been propounded in this text, it is apparent that subjectivity predominates the low back pain syndromes and pain is the major concern of both the patient and the therapist.

The modalities of treating acute pain with the interruption of the nociceptive pathways has been addressed in numerous texts and summarized by the author.<sup>27</sup> These modalities and approaches will be summarized here as related to the low back but another concept has emerged that is proving to be more effective.<sup>28-30</sup>



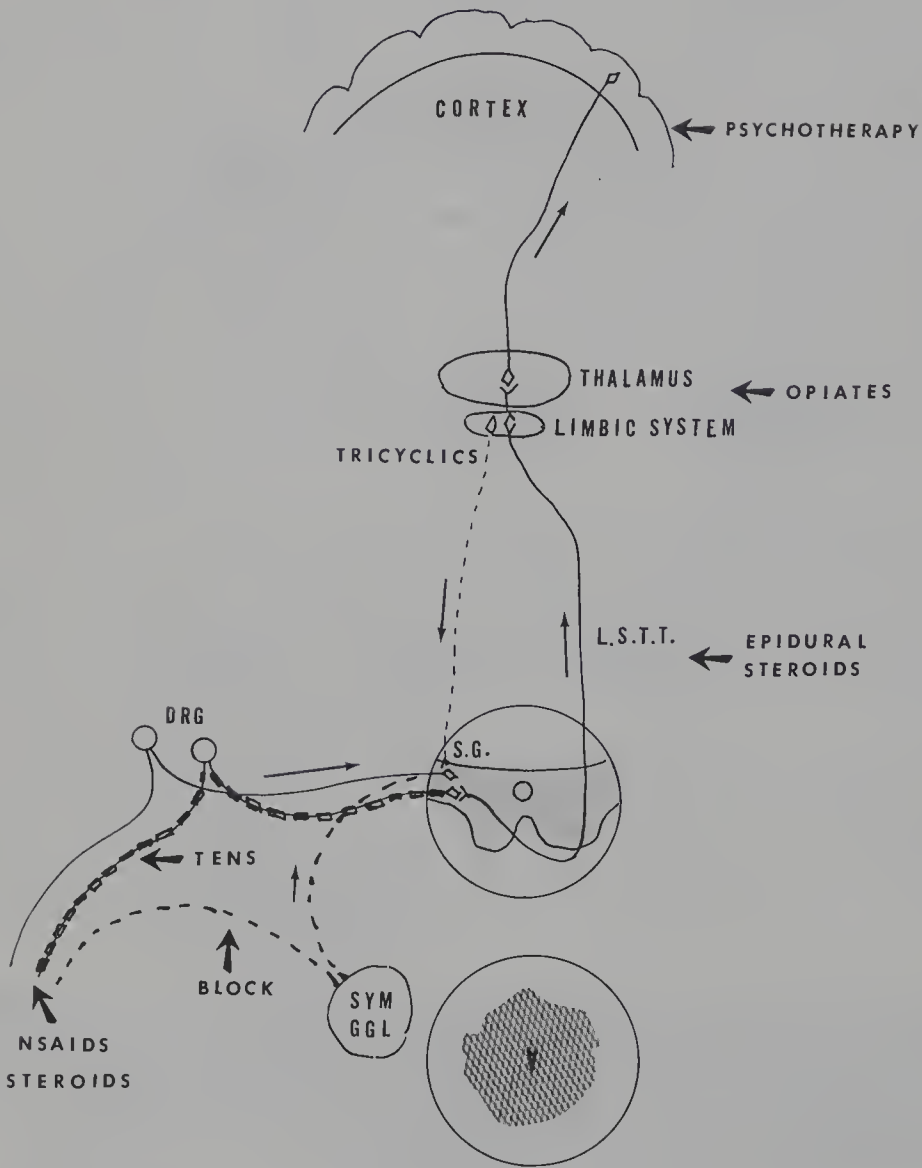


Figure 2-13. Sites of action in the treatment of pain. In the neural transmission of nociceptors, intervention occurs at several sites. At the periphery, the dorsal root ganglion (DRG) transmits impulses into the substantia gelatinosa (SG). Steroids or NSAIDs intervene in this pathway. TENS (transcutaneous electrical stimulation) also intervenes at this site as does a nerve block of sympathetic impulses. Epidural steroids intervene at the lateral spinothalamic tract level (LSTT) and tricyclics at the limbic system and the thalamus. Opioids intervene at this level also. Psychotherapy intervenes at the cortical level.

The concept of pain as a purely sensory phenomenon has shifted to the understanding of pain as a perceptual process.<sup>31</sup> The current medical model of automatically presuming chronic pain occurring from a medical and psychological viewpoint was challenged by Fordyce, et al., who proposed an operant behavioral model.<sup>32</sup> This model addressed function rather than pain as the target.

Unlike the current medical model of treating the pain, Johnson and Fordyce found that there is "not a corresponding significance of illness and/or injury or the degree of impairment to the amount of pain complained by the patient." In fact, "physical findings accountable for the pain are often absent."<sup>33</sup> Chronic pain has been accepted as "pain that persists after healing is known to have taken place," as Loeser says.<sup>34</sup>

*Pain* is currently defined as a perceptual response which may or may not have had a nociceptive antecedent incidence. Pain is a private experience that can only be appreciated by others through verbal or behavioral activities by the sufferer. Increased attention and nurturing by others only reinforce the pain behavior.

Patterns of guarding or overguarding lead to chronicity not only in perception but by addition of disuse.<sup>35</sup> The subjective nature of the history, which depicts the disability and the absence (often if not usually) of objective findings leading to a discernible impairment, leads to a nondiagnosis on which is based a treatment protocol. The current innovation of approaching low back pain with the sports medicine early care concept is consistent with addressing function and not the relief of pain.

In this concept prolonged bed rest, inactivity, and passive modalities are condemned. Long-term bed rest has debilitating effects on muscle strength and flexibility, bone density, and enforced disability with lack of patient involvement in the healing process. Deconditioning and debility result from prolonged inactivity.<sup>35</sup> Disk integrity has also been jeopardized from inactivity.<sup>36</sup>

Initial rest of the injured part is physiological, as is the use of modalities such as ice, heat, ultrasound, and even massage.<sup>37,38</sup> While resting the injured part, the remainder of the body must remain active for cardiovascular, metabolic, and psychological reasons.

Education remains paramount in acute care and rehabilitation. It would seem redundant to state that informing the patient in understandable terms, concepts, and anatomical principles is predominant in any treatment protocol. Physicians and even therapists are so informed in the use of medical terms that they fail to remember that most patients are not so educated and knowledgeable. Terms can be constructive or destructive, depending on how they are used and explained. *If the patient understands, the patient participates in the recovery process.*

The use, misuse, and inappropriate use of words has a significant effect on a patient in pain. The fear of the unknown is no greater than it

is in this condition. Explanation of every test and its significance is mandatory. The basis for the treatment and its goal is also mandatory. The normal expected aspect of the symptoms and the resultant disability must be clarified and the prognosis explained in meaningful terms. The fears of the patient caused by misinformation gained from television, magazines, well-meaning friends, and so on must be accepted and overcome.

The statement that "the doctor does not have time to explain" is invalid. No explanation given by the physician and the therapist, to the patient is too time consuming. The truth may well be that most doctors do not have the ability or the vocabulary to clearly communicate with the patient in giving a meaningful description of the condition and the cause of the symptoms.

Work is often not specified as it relates to restrictions and rewards. Acute pain has now been superseded by chronic pain in the evaluation of the patient with pain.<sup>2</sup>

## CHRONIC PAIN

Enforcement and reinforcement are the bases of operant conditioning.<sup>32</sup> Attention to a complaint enforces and reinforces the intensity of the reaction and ensures a recurrence if not a persistence. Reinforcements are termed either *positive* or *negative* in the concept, with the former encouraging pain behavior and the latter diminishing or removing it.

Positive reinforcement is beneficial and a pleasant reward for the complaint.<sup>39</sup> Positive reinforcement can be sympathy, attention, monetary gains, and release from demands otherwise unmet. A negative reinforcement is when a behavior does not elicit something beneficial or pleasant but rather invokes disregard, avoidance, neglect, or rejection. This negative response prevents recurrence and minimizes chronicity.

Acute treatment done appropriately for a reasonable time and carefully monitored and evaluated does not necessarily become a positive reinforcer. The opposite, prolonged, inappropriate, and meaningless treatment, becomes a positive reinforcer with dire circumstances such as recurrence and continuation into chronicity.

As Sanders states, "Pain experience involves overt behavior."<sup>40</sup> There are various factors involved, including the stimulus intensity and the patient's pain threshold, the integrity of the central nervous system, and the patient's previous experience, by which the significance of the pain is judged.

In essence the pathological condition, based on severity of the incidence and the tissue response (pathology), causes a response (pain),

with the latter influencing the former. The initial therapeutic response is to minimize or eliminate the nociception with expected resultant diminution of the response (pain behavior).

Chronic pain occurs when there is prolonged pathology or prolonged pain behavior. The former may exist and needs discovery and physical attention. The latter may persist even when the former has subsided or disappeared. Reinforcers are considered to play a large role in this chronic pain behavior concept.

Practically all pain behaviors are operants.<sup>41</sup> In consequence, even in the presence of persisting albeit diminishing pathological stimulus, the pain behavior may persist. Behavioral treatment methods are not intended to treat but are intended to prevent excessive disability and minimize the expression of suffering while normal healing occurs and function remains or returns.

Treatment (operant) concepts intend to (1) decrease verbal and overt pain behavior, (2) decrease consumption of pain medication, (3) increase physical activity, and (4) alter the undesirable environmental effects.

Decrease of verbal and overt behavioral actions is attempted by withdrawing social reinforcers (attention) and avoiding verbal acknowledgment of pain. Information as to function and testing is appropriate, even necessary, to monitor the condition, but this does not evaluate the degree of pain.

Consumption of pain medication is controlled by avoiding the prn prescription, which is dependent on the patient's request as determined by their evaluation of the severity of the pain. Medication should be given in a regulated dosage and frequency *in an unrecognizable vehicle*. The dosage can be regulated and gradually diminished until eliminated. The pain is recognized and accepted in the acute stage and adequately treated. As the condition is monitored, the expected pain is appreciated and so treated.

The third behavior is function and its diminution by the pathology and the resultant disability. The health care provided encourages increasing and recorded activity, which acts as an objective measurement to the therapist and, more important, to the patient.

Rest has always been the indicator for the degree of pain in the current medical model. Rest is actually a reinforcer. Objective measurement of rest becomes an indicator of activity. It decreases in time and frequency as activity increases.

The effect on the environment relates to the reaction of the employer, the spouse, and the family. All can play roles as reinforcers: positive or negative.

Cognitive approaches to low back pain must be understood as not implying that the pain is not real but merely conceived. Epictetus said,



"Men are disturbed not by things but by the views they take of them."<sup>42</sup> Chronic low back pain has several components besides pain. They are anxiety, fear of recurrence, and depression.

The cognitive-behavioral perspective of pain management of the patient with chronic low back pain is primary in today's understanding and must be addressed early during the acute phase.

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## CHAPTER 3

# Examination of the Patient with Low Back Pain

A patient presenting with symptoms of pain in the low back must be evaluated as having merely low back pain, having leg pain, or having both back and leg pain. Also the history is more pertinent than is the examination, but the examination, performed adequately, informs the examiner as to the structural basis of the impairment, the tissue site, and thus the mechanical basis of the complaints.

A rational diagnosis of low back syndrome is handicapped by the lack of reliable objective tests.<sup>1</sup> Pain is the basis of low back symptoms and thus a nociceptive site must be identified after a careful history has determined the mechanism of the factors irritating these tissues. The tissue sites of potential nociception within a functional unit of the lumbar spine have been specified and can be summarized as:

1. External fibers of the intervertebral disk annulus
2. Posterior longitudinal ligament
3. Nerve root dura
4. Synovial capsular tissue of the facets
5. Ligaments: interspinous, supraspinous, or both
6. Erector spinae muscles (and their fascia)

Most mechanical pain can be simplistically related to variants of discogenic or myogenic pain.<sup>2</sup> Additionally, however, there are other tissues such as the facets, ligaments, and the dura of the nerve roots that need consideration.

## TECHNIQUES OF EXAMINATION

The examination begins by observing the patient's facial expression, attitude, posture, and manner of mobility. The emotional tone of giving the history is also to be observed. The manifestation of pain is elicited in these aspects of the examination. The experience of pain is often transmitted to the observer by the facial expression and tone of voice, as pain is a subjective experience and its manifestation must be transmitted externally. The facial expression, verbal descriptions, tone of voice—from plaintive to angry—and overt mannerisms all depict the presence of pain: its quality, intensity, and even its relationship to tissue site.

Understanding the impairment is the objective of the physical examination, but the determination of the resultant disability must also be established from the examination as well as from the history. *Impairment* has been defined by the World Health Organization as “any loss or abnormality of psychological, physiological, or anatomical structure or function.”<sup>3</sup> This can be increased to include “leading to loss of normal bodily function.”<sup>4-6</sup> Social security guidelines have defined *impairment* as “being demonstrable by medically acceptable clinical and laboratory diagnostic techniques.”<sup>7</sup>

Clinical assessment of impairment should therefore be based solely on reliable objective clinical signs that are clearly separable from cognitive, psychological, or behavioral features of the illness. The impairment identified must specifically relate to the disability claimed or demonstrated by the patient.

All the motions or actions of the patient depict the disability and indicate to the examiner how the patient hurts and to what extent and what movements the patient finds painful and how the pain impairs function.

The disability is essentially what the patient cannot do or does but with pain. The objective reasons for that disability should be the impairments that the examination reveals.

The gait reveals any impaired motor function of the legs and the trunk. A limp must be analyzed as to its mechanism and basis. Complaints of pain during gait focus the specific time and therefore the basis of the limp as declared by the patient. The observer can only analyze the gait and judge from the evinced grimace and verbal complaint that pain is elicited.

Gait abnormalities reveal motor weakness of involved muscle groups<sup>8</sup> such as a drop foot, steppage gait, trunk asymmetry, or even pain on weight bearing. Abnormal gaits that are created by fear of pain

or that depict the disability experienced must be carefully evaluated as being expected from the ultimate findings, exaggerated or unphysiological. Appropriate neurological examination of the foot, ankle, and lower leg will reveal the basis for the limp. The neurological examination will be discussed.

Sitting or arising from the seated position must be observed as to trunk physiological movements and leg weakness. Pain must be evaluated as occurring during the sitting, after being seated, and on arising. There are physiological movements of the lumbosacral spine that occur during these activities, and they must be analyzed.

During sitting, the spine normally assumes a kyphotic posture, which means trunk flexion. If trunk flexion is painful and limited, that can be observed. The erector spinae muscles decelerate the flexion, and when restricted in elongation, they diminish this effort.

Once the patient is seated, the sitting posture must be observed. Physiological sitting requires some reversal of the lordosis and central sitting without functional scoliosis (Fig. 3-1).

The history has revealed what actions, movements, and positions have resulted in pain and limitation. The examination is the attempt to ascertain which tissue is now the causation of the pain. During the examination the purpose should be to reproduce the precise pain complained of by the patient. This clearly depicts the mechanism of the evoked discomfort and indicates the tissue responsible. This attempt from the examination is to reproduce the subjective symptoms and is not significantly objective.

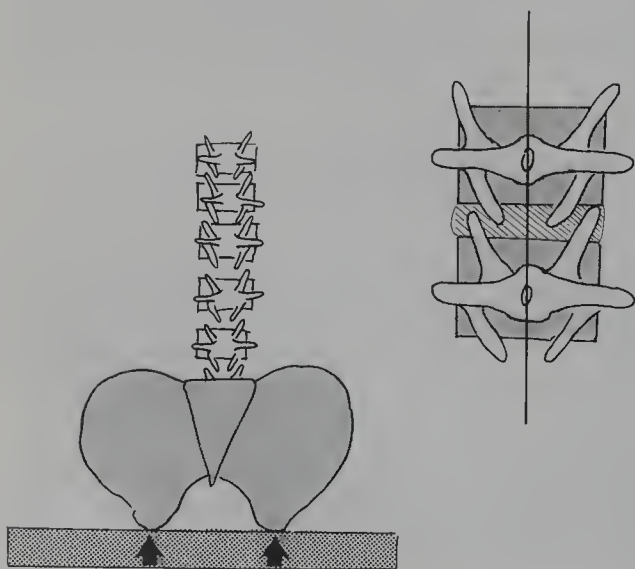


Figure 3-1. Level seated pelvis and erect spine. The level pelvis should bear weight equally on both ischial tuberosities (arrows in left figure) making the pelvis level. Thus the spine is erect unless there is a structural scoliosis. The individual function unit (right figure) has both endplates parallel, the disk symmetrical, and the facets properly aligned.



The principal sources of pain are (1) injury to the supporting soft tissues, especially ligaments, muscles, and tendons, (2) injury or inflammation of the posterior joints (facets), and (3) injury to the disk structures.

Injury to soft tissues such as ligaments is rarely massive such as occurs in knees, shoulders, and other extremity injuries. Ligamentous injuries in the spine are usually secondary to overload, inappropriate movements, or external forces.

Muscles tear more readily than do tendons or ligaments. Muscles also tear more readily in eccentric contracture (lengthening while under tension), which (as discussed in Chapter 1) is a frequent muscular function of the spine in bending and lifting. Tears, when they occur, do not occur within the midsubstance of the muscle but at their myotendinous junction.<sup>9</sup> Residual pain may result from the tendons to the bony attachments of these injured tissues.

Inflamed muscles may undergo compartment syndrome, in which there is swelling within an inflexible compartment (fascia) with resultant ischemia. There is little or no evidence that compartment syndrome occurs in the erector spinae muscle groups.<sup>10,11</sup>

Another type of symptomatic muscle soft tissue injury that has been associated with low back injuries and their sequelae has been termed fibromyalgia or fibrositis. This condition consists of generalized fatigue, stiffness, and numerous tender spots termed *trigger points*. The mechanism of this condition is currently unestablished and its relationship to trauma questioned as it has been produced by mere sleep deprivation.<sup>12</sup> Of interest is that fibromyalgia symptoms can be eliminated by fitness training more rapidly than by local approaches to the trigger points.<sup>13</sup> As fibromyalgia is currently considered to be a neurological reflex abnormality rather than a structural tissue injury, its relationship to trauma remains valid.

Myofascial pain as a cause of low back pain must adhere to the criteria of myofascial disease, which are sleeping disturbances, generalized muscular pain, and the presence of palpable trigger points in the affected muscle. As most myofascial pain syndromes affect the trapezius and neck shoulder areas, low back manifestation is infrequent as a sole symptom.

Connective tissue injuries, as these have been postulated to be, repair slowly as they have a deficient blood supply. As their role is tension resistance, repair demands production of new collagen fibers. This requires diffusion from adjacent tissues, thus activity rather than inactivity is mandatory for repair. Activity may be gentle passive or gentle active as pain permits.



## EXAMINATION OF MUSCLES AND TENDONS

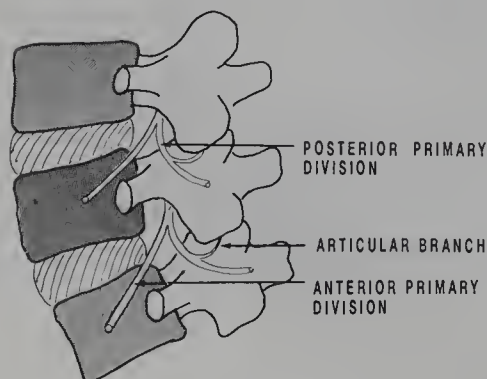
Muscle pain is accompanied by muscle tenderness that is palpable by the examiner but the response by the patient is subjective as to its presence and its severity. Pain can also be elicited by passive stretching but this motion also involves the other periarticular soft tissue. Pain can be reproduced by actively contracting the involved muscle, but this active contraction also increases intradiskal pressure so is not an isolated tissue response.

There are no laboratory or imaging studies that confirm myogenic pain, so subjectivity predominates.

## FACET PATHOLOGY AND EXAMINATION

An entire chapter has been devoted to the zygapophysial (facet) joints. Consideration here is the examination confirming or at least alluding to these joints being the cause of the alleged pain.

Badgley first confirmed that abnormalities within the facet joints could be a source of persistent (chronic) pain.<sup>14</sup> He postulated the innervation of the capsule and periosteum of the facets as being the medial branch of the posterior primary ramus (Fig. 3-2). Pain emanating from the soft tissues of the low back had been identified by Inman<sup>15</sup> in 1944; however, subsequent studies have revealed that numerous overlapping neurological innervations to these spinal structures occur as well as overlapping of somatic nerves with autonomic nerves.<sup>16,17</sup> This latter innervation also relates the pain as being, therefore, influenced by the emotions: anxiety and depression.



**Figure 3-2.** Innervation of the facets. Upon emergence from the foramen, the nerve roots divide into anterior primary and posterior primary divisions. A small articular branch emerges that is sensory to the facets.

Percutaneous injection of a local anesthetic was assumed to relieve pain from the facet joints,<sup>18</sup> but the multiple innervations in that area invalidated the facets as being the primary site of pain. Injection of a hypertonic saline solution into the joint also created low back pain, but these were not radiographically confirmed.<sup>19</sup> Repetition of this study under radiological localization confirmed the facet joints to be sites of nociception.<sup>20</sup>

The amount of injected hypertonic saline increased the area of pain, dependent on the amount injected. A small amount caused pain into the buttock, and as the amount increased, the pain was referred down the posterior thigh and the calf. No skin sensory abnormalities were noted from these studies, but radiating pain from the facets was confirmed.

Clinically confirming the facets as *the* site of pain is not so clear. No clear diagnostic examination procedures can clearly ascertain the facets as being responsible. Local tenderness over a facet has been described as has lateral manipulation via the posterior spinous process.<sup>21</sup> Hyperextension of the lumbosacral spine combined with lateral flexion allegedly impinges the unilateral facets on the concave side, yet this test, also, is subjective.

As this procedure also influences intradiskal pressure changes, it is not specific for facet pain. Only intra-articular injections are reasonably confirmatory.

## EXAMINATION OF LUMBAR DISK PAIN

The intervertebral disk has veritably become the *bête noire* of low back pain since the classic work of Mixter and Barr in the 1930s.<sup>22</sup> The intervertebral disk is the largest avascular tissue in the body and receives its nutrition via imbibition from surrounding tissues. As the nucleus is furthest from its blood supply, it has the poorest nutrition.

The hydrodynamics of the disk have been intensively studied. Gravity affects the disk, as does spinal muscular contraction. Approximately 3% to 10% of the fluid is lost during an average day and requires 10 hours for total loss and 2 hours for recovery.<sup>23</sup> As nutrition is dependent on imbibition, cyclic loading is therapeutic. An entire chapter is devoted to this subject. Here, we will ascertain just the pertinent aspects of the initial clinical examination.

### Objective Signs Elicited in an Examination

The ideal would be to determine what objective signs relate to disability. Following an acute injury when the involved tissues are

inflamed, physical signs can be elicited. When the acute low back pain becomes chronic, the story changes. *There are no objective signs that have been ascertained as verifiable in establishing the basis of disability in chronic low mechanical back pain.*<sup>24</sup>

In patients with low back pain *and* leg pain, objective signs are more meaningful and diagnostic even though they, too, do not always clearly and totally relate to the subjective disability claimed by the patient.

## STANDARD PHYSICAL EXAMINATION TESTS

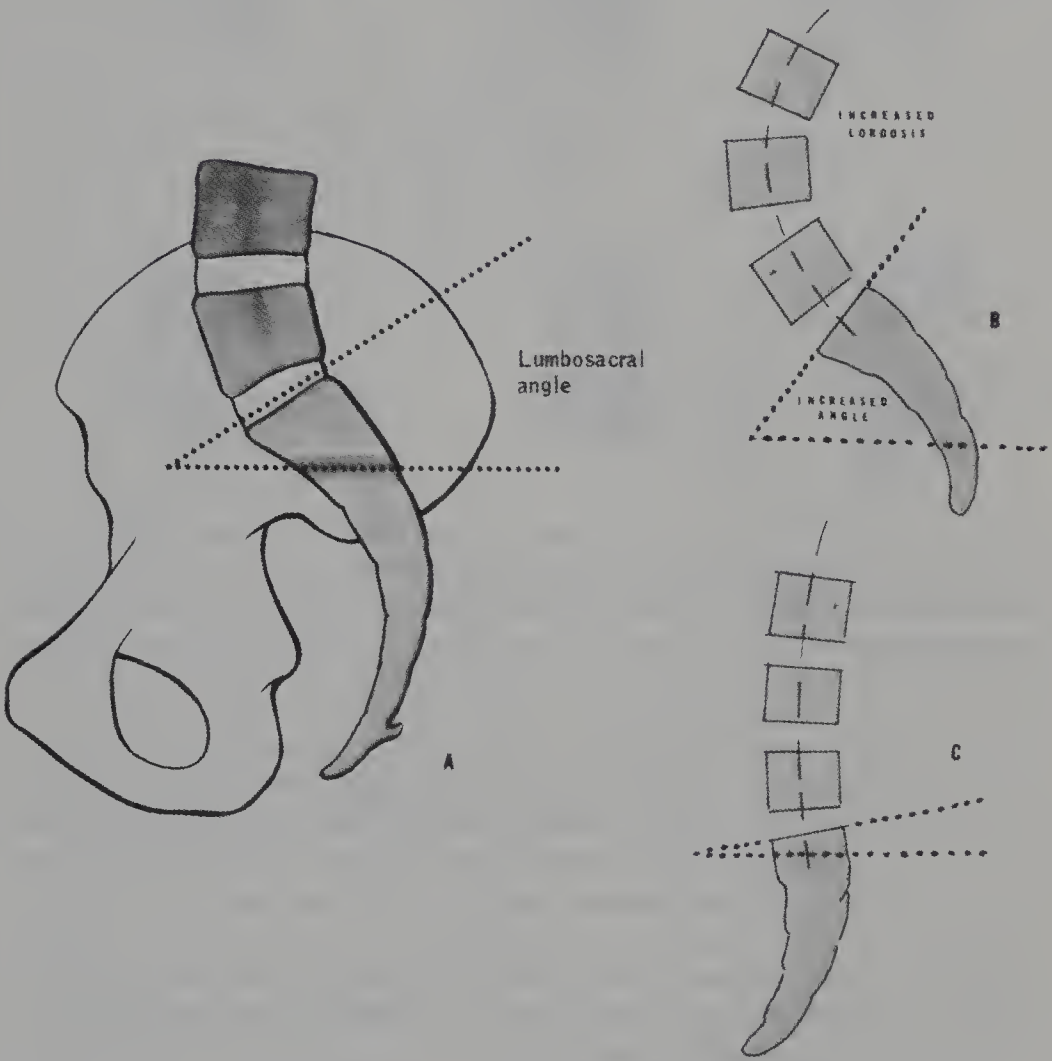
The standard tests performed today to evaluate the suspected impairments as causing the disability can be listed as follows:

1. Excessive (abnormal and symptomatic) lordosis
2. Lumbar kyphosis
3. Lumbosacral (functional) scoliosis
4. Sacral angle
5. Lumbar flexion: range of motion (ROM)
6. Lumbar extension (sagittal and with lateral flexion)
7. Lateral flexion
8. Straight leg raising (SLR) or Lasègue test
9. Contralateral SLR
10. Tenderness: interspinous, paraspinous, buttocks or lower extremity
11. Strength testing: trunk flexors, trunk extensors, lower extremities
12. Precise pain reproduction

Each test will be defined and illustrated and its value discussed.

### Lordosis

Excessive lordosis has been advocated as the major cause of postural pain, prolonged functional position, facet pain, and even radiculopathy. This lordosis implies an alteration of the sacral angle (Fig. 3-3), causing low back pain and, when associated with leg pain, a possible radiculopathy from alteration of the foraminal opening (Fig. 3-4) or possible herniation of the disk. Accurate measurement of the sacral angle, considered to be responsible for excessive lordosis,<sup>25,26</sup> remains controversial.<sup>18</sup>



**Figure 3-3.** Lumbosacral angle. The lumbosacral angle is measured from a line parallel to the superior aspect of the sacrum and a horizontal line (*dotted lines*) (A). If the angle is increased (B) the superincumbent spine curves appropriately and the lordosis decreases (C) when the angle is smaller.

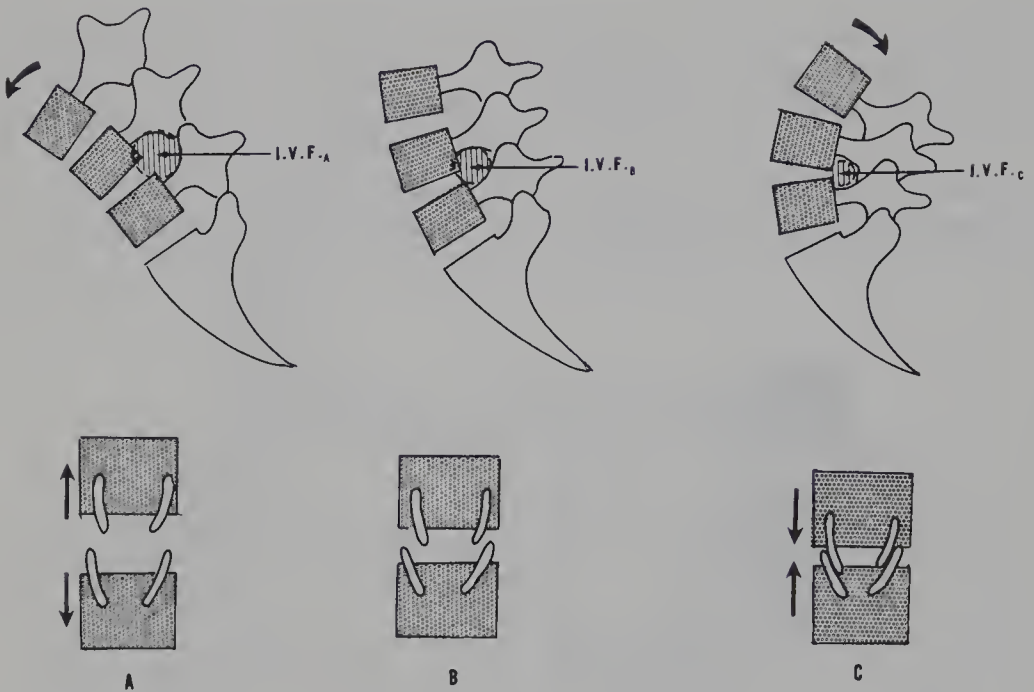


Figure 3-4. Facet movement in flexion and extension: foraminal opening. The facets separate on trunk flexion with opening of the foramina (A). The neutral position of the lumbosacral spine (B). On extension the facets approach and the foramina narrow (C).

The sacral angle implies the concept of pelvic tilt, as the sacrum is firmly attached to the pelvis, which rotates about the hip joints (Fig. 3-5). The manner of determining pelvic tilt is merely a visual observation and varies with the examiner and to when and how the patient is examined. It is an inaccurate and subjective measurement not dependent on instrumentation. Pain elicited from passively or actively accentuating the lordosis is subjective but does implicate the movement as causative of the patient's symptoms.

Posture generally implies evaluation of the spine as viewed from the side.<sup>27</sup> Good posture implies an energy-economical cosmetically acceptable stance with no incurred pain or discomfort. All superincumbent curves: the sacral, lumbar, thoracic, and cervical are balanced against the center of gravity.

Effortless erect postural stance is accomplished by:

1. Intradiskal pressures within adjacent vertebral endplates
2. Tension of the intervertebral annular fibers
3. Pelvic angulation as determined by the attached muscles and ligamentous structures



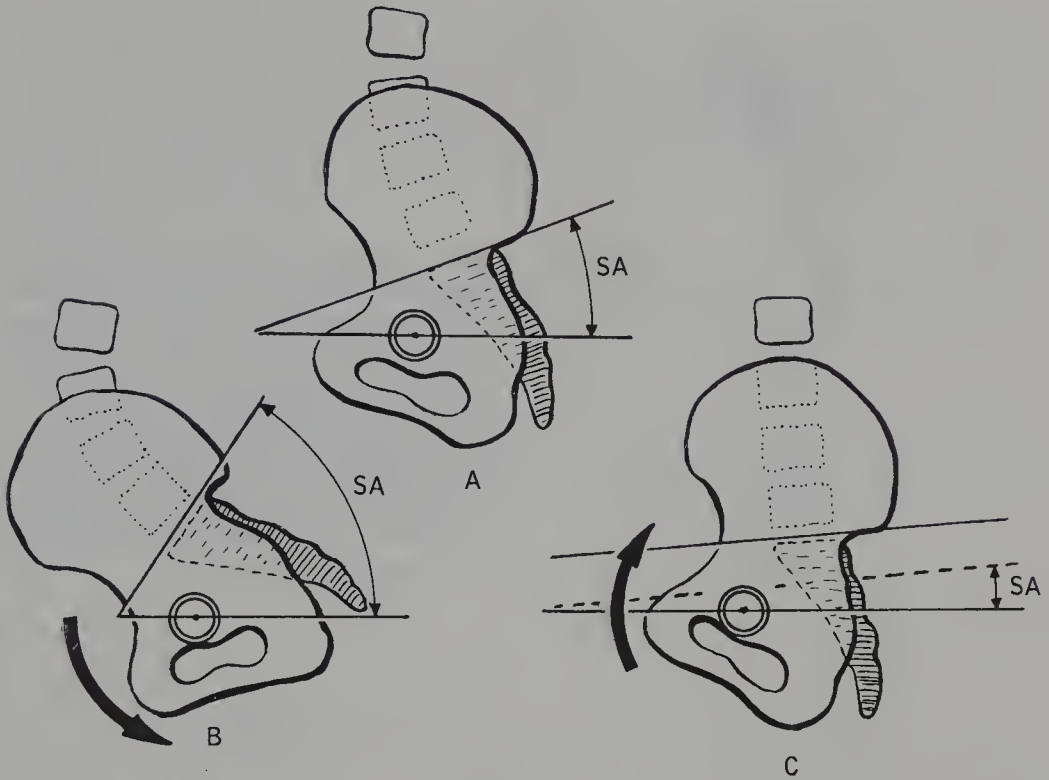


Figure 3-5. Lumbar angle and pelvic tilt. The term *pelvic tilt* is commonly used. Rotation of the pelvis from the neutral position (A) shows the position of the sacrum, which is firmly attached to the pelvis. The sacral angle (SA) is a physiological measurement. As the pelvis rotates anteriorly downward (B) (curved arrow) the angle of the sacrum changes. In posterior rotation (C) the sacrum assumes a more vertical alignment with a smaller sacral angle (SA) and in anterior rotation (A) the sacral angle is greater.

There are predominant influences on the posture that must be recognized:<sup>28-30</sup>

1. Familial or hereditary factors
2. Structural anomalies, either congenital, acquired, neurological, muscular, or skeletal
3. Postures of habit or training during the developmental years and acquired from prolonged occupational stresses

The statement of Waddell et al. that “posture is completely independent of behavioral signs”<sup>24</sup> is not accepted by the author, as posture is body language (Fig. 3-6) that depicts the emotions and excessive lordosis is not exempt.

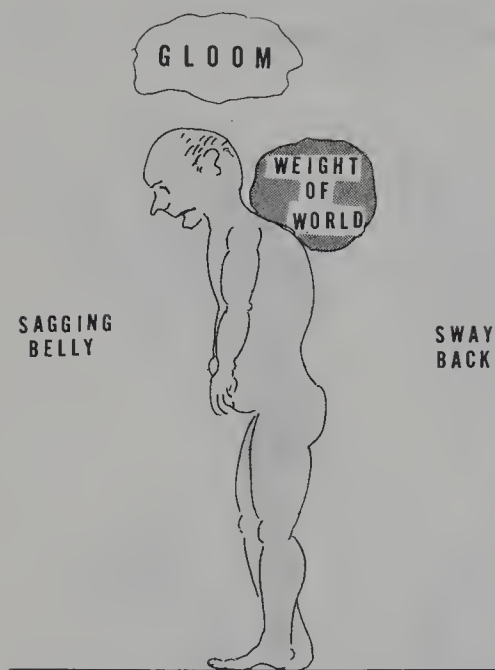


Figure 3-6. Postural depiction of the emotions. The depressed person portrays the emotions with an increased dorsal kyphosis (carrying the weight of the world on one's back), an increased lumbar lordosis, and a sagging abdomen. The mood is gloom. (From Cailliet, R: *Understand Your Backache*. FA Davis, Philadelphia, 1984, with permission.)

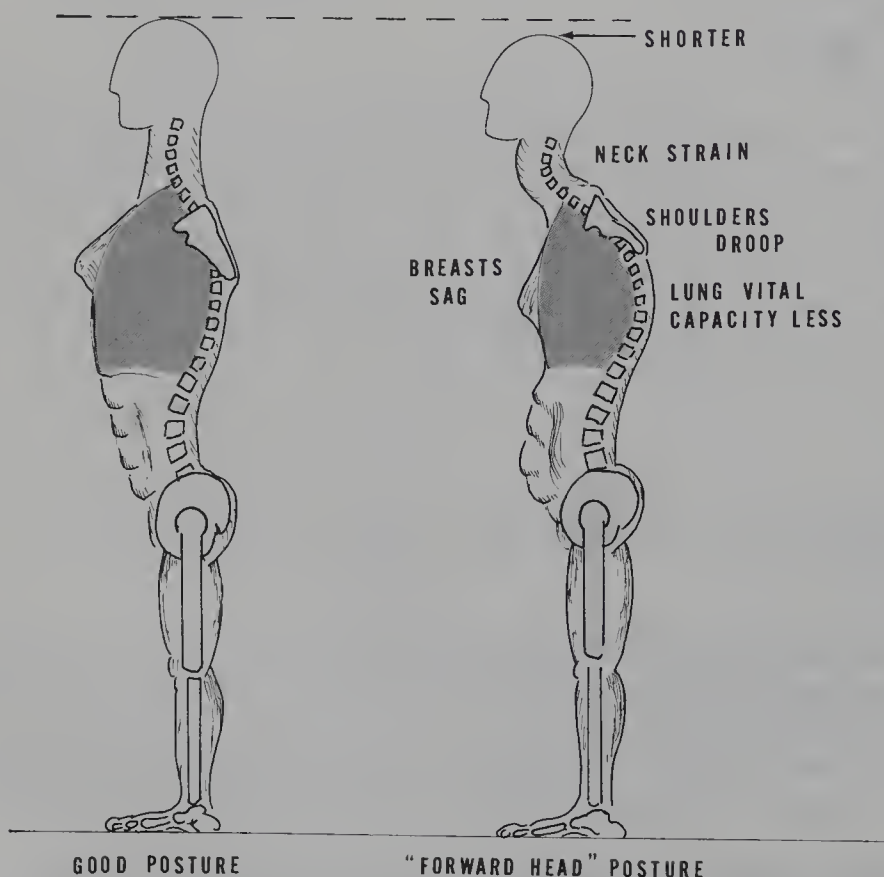


Figure 3-7. Good posture versus forward head posture. Good posture (left) is compared with forward head posture (right) and all of its sequelae: sagging breast, impaired lung capacity, drooping shoulders, and neck strain.

Good posture versus forward-head posture can also be visualized and all the tissue reactions revealed (Fig. 3–7). That abnormal posture, involving the lumbosacral spine, causes pain remains unconfirmed<sup>31</sup> yet reproducible. Symptomatic hyperlordosis during the examination may well be a factor in low back pain (Fig. 3–8) and be elicited in the history (Fig. 3–9).

The lordosis of pregnancy and wearing high heels have been postulated as causes of low back pain in women (Fig. 3–10), but these as causes of the condition are remedied when pregnancy terminates and high heel shoes are removed.

Postural correction from altering the height of a chair, using a footstool, a low back pillow, or both (Fig. 3–11) can be diagnostic as well as therapeutic. The sitting lordosis and its modification is visible during an examination.

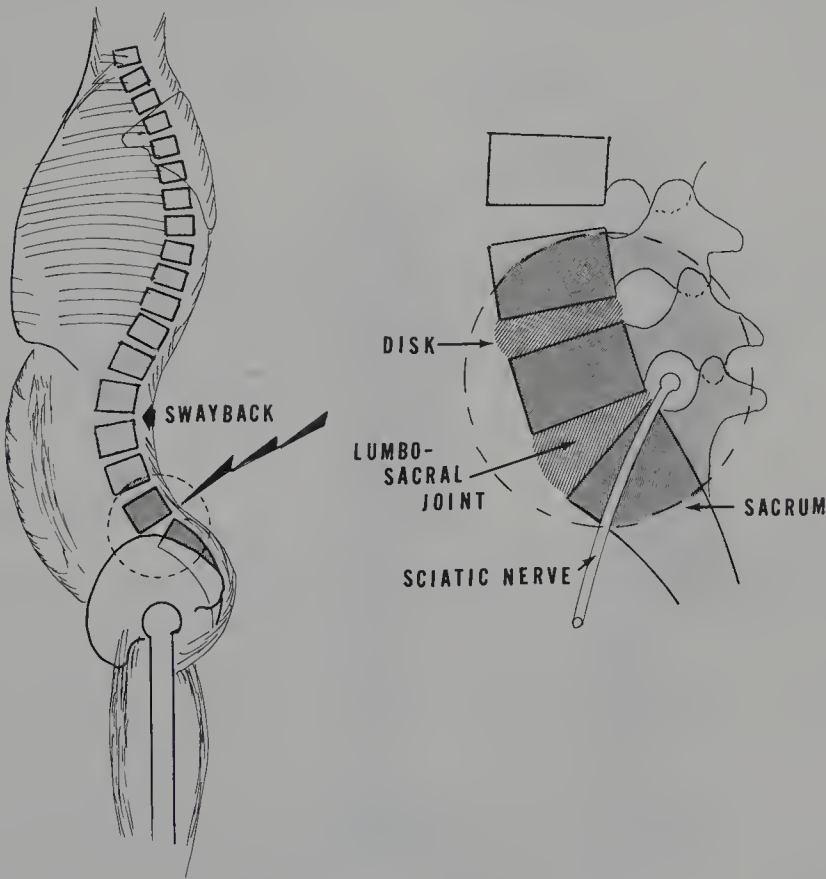


Figure 3–8. Mechanisms postulated in symptomatic lordosis. The increased lordosis termed *sway back* causes an increase in lumbosacral angle causing closure of the foramen between L5 to S1 compressing the sciatic nerve. The lumbosacral joint is between fifth lumbar vertebra and the sacrum with the intervertebral disk shown.

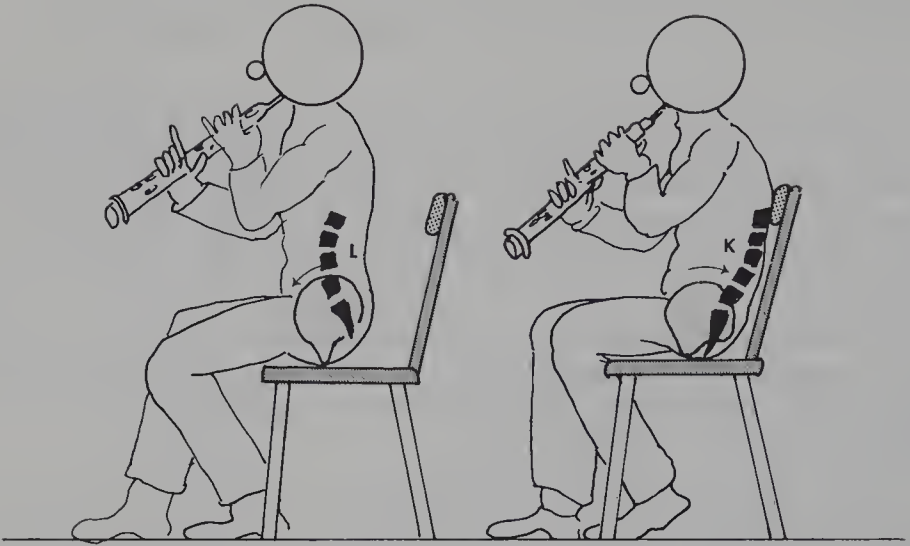


Figure 3-9. Postures in a performing artist. Figure to the left shows forward rotation of the pelvis (*arrow*) with a slight increase in lordosis (L). The artist on the right shows posterior rotation of the pelvis with decrease in the lordosis: a lumbar kyphosis (K). Either position can be symptomatic if excessive and prolonged.

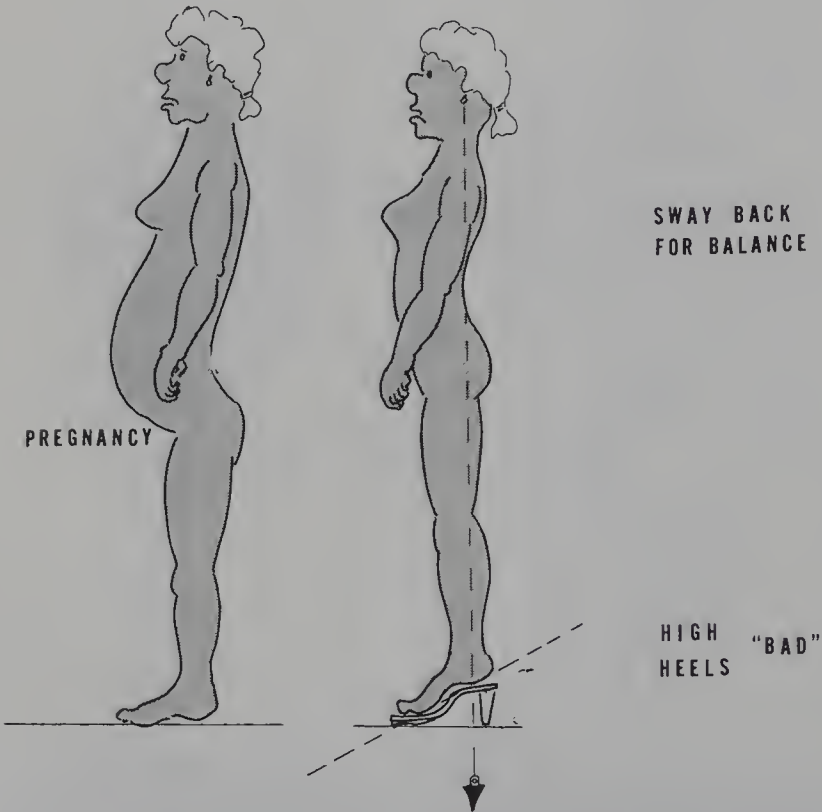
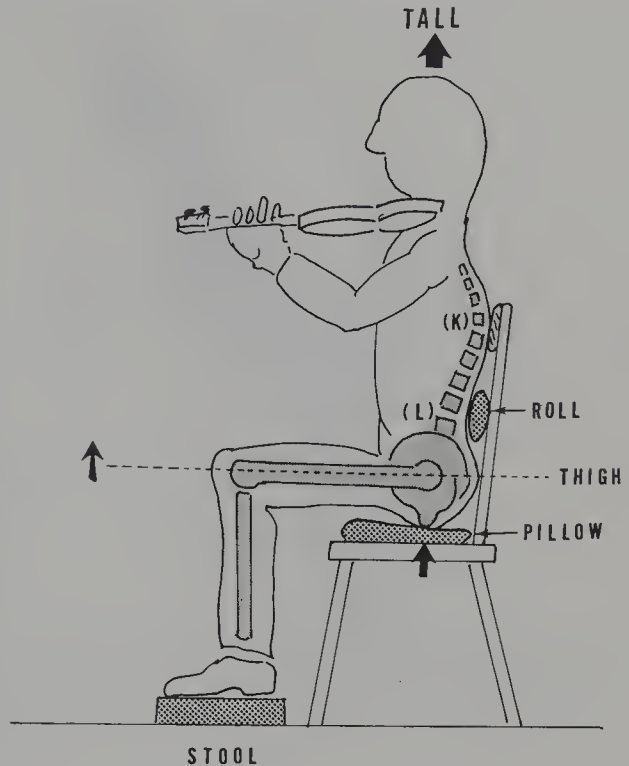


Figure 3-10. Lordosis in pregnancy and from wearing high heels. Symptomatic excessive lordosis from pregnancy is shown on the left and from wearing high heels on the right.

**Figure 3–11.** Postural corrective measures in sitting. When a sitting posture has been found to be symptomatic from either excessive lumbar lordosis or prolonged kyphosis several corrective measures can be considered. Sitting tall may relieve the low backache as may a stool under the feet, a sitting pillow, and a roll behind the lumbar spine to correct the lumbar lordosis (L) and diminish the dorsal kyphosis (K). All these measures assure that the femur is horizontal (dotted lines).



Excessive lordosis as *the* cause of low back pain cannot be objectively confirmed, but the effects of lordosis can be modified by manipulating the posture both in standing and sitting and by having the patient claim the benefit or aggravation.<sup>32,33</sup> This is a valid aspect of a meaningful examination (see Fig. 3–12).

## Lumbar Kyphosis

Reverse of the physiological lordosis has been given much attention as to being causative of low back pain (Fig. 3–13).<sup>32,33</sup> Reversed lordotic posture causing low back pain is attributed to erector spinae muscle elongation, posterior spinous ligamentous strain, or posterior nuclear protrusion with central pressure on the posterior longitudinal ligament.

Prolonged sitting with excessive kyphosis allegedly causes posterior bulging of the disk nucleus (Fig. 3–14) with elongation of the posterior annular fibers that gradually tear the annular fibers, permitting the nucleus to internally herniate. The resultant posterior annular bulge presses on the posterior longitudinal ligament, which is well innervated by nociceptive fibers. Repeated flexion or kyphosis, therefore, is also considered as causing this kyphotic painful condition.



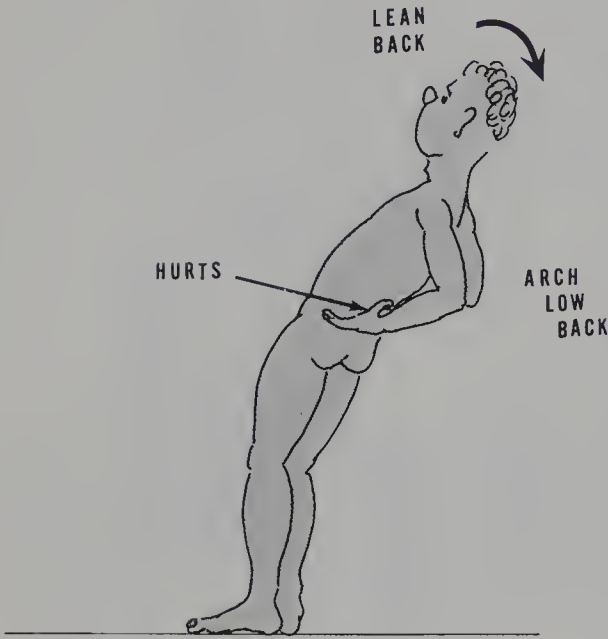


Figure 3-12. Confirmation of lordosis causing backache. In the clinical setting the complaint of low backache considered as caused by excessive lordosis can be reproduced by causing the patient to excessively lean back (arrow) at the low back region.

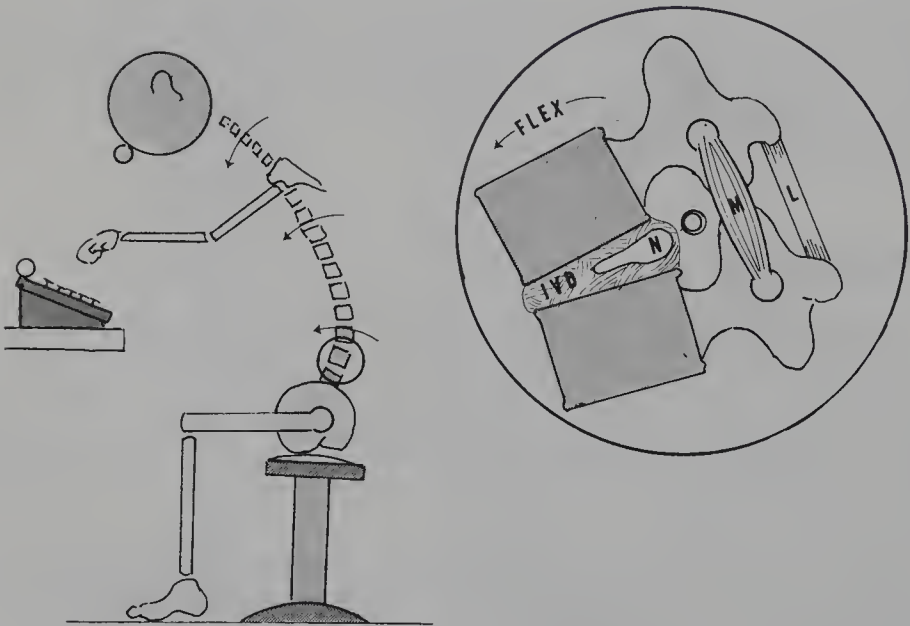


Figure 3-13. Prolonged sitting posture. The kyphotic lumbar posture replaces strain on the erector muscles (M), the supraspinatus ligaments (L), and the posterior annular fibers. The nucleus (N) of the intervertebral disk (IVD) protrudes posteriorly to compress the posterior longitudinal ligament (not marked) against the nerve root.

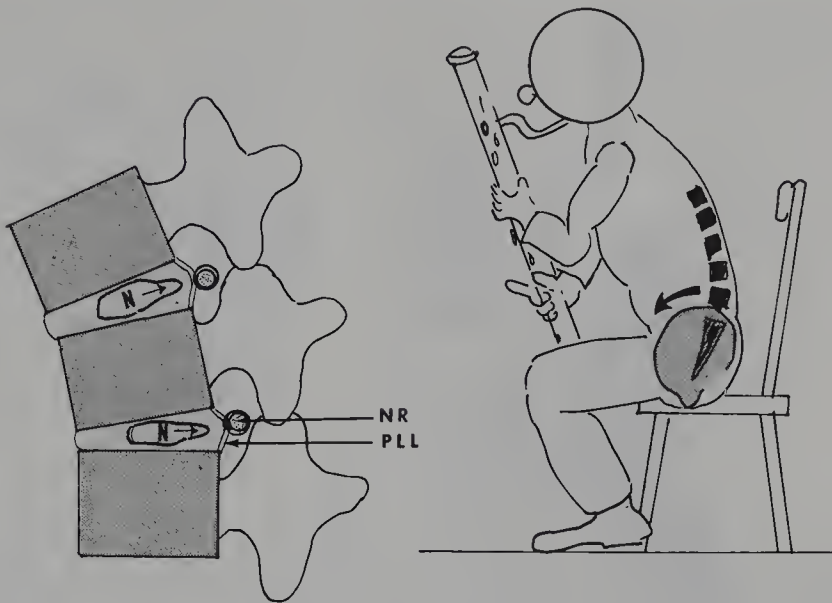


Figure 3-14. Prolonged sitting causing posterior disk bulge. The kyphotic lumbar posture flexes the spine causing the nucleus (N) of the intervertebral disk to protrude posteriorly to press against the posterior longitudinal ligament (PLL) against the nerve root (NR).

Clinically a central disk protrusion against the posterior longitudinal ligament is considered when the patient, in attempting to forward flex, is limited and the last few degrees of flexion cause low back pain. Nuchal flexion at the endpoint of trunk flexion then usually aggravates the pain. Being central, there are no leg symptoms.

Objectively confirming this fact, however, requires confirmation by computerized axial tomography (CAT) or magnetic resonance imaging (MRI), done with the spine fully flexed. Merely reproducing the low back pain with sustained flexion is not objectively discernible as the pain elicited is subjective and the degree of kyphosis not measurable.

## Lateral Pelvic Tilt

Accurate measurement of lateral pelvic tilt and uneven iliac level are clinically verifiable, but accurate measurement is difficult. Causation of low back pain from this is also subjective. Pain claimed by the patient and noted and reproduced by the examiner is a subjective experience (Fig. 3-15).

Leg length and commensurate pelvic tilt can be measured (Fig. 3-16).<sup>34</sup> Scoliosis (Fig. 3-17) can also be visualized, but its relationship to low back pain is only assumed.<sup>35-37</sup>

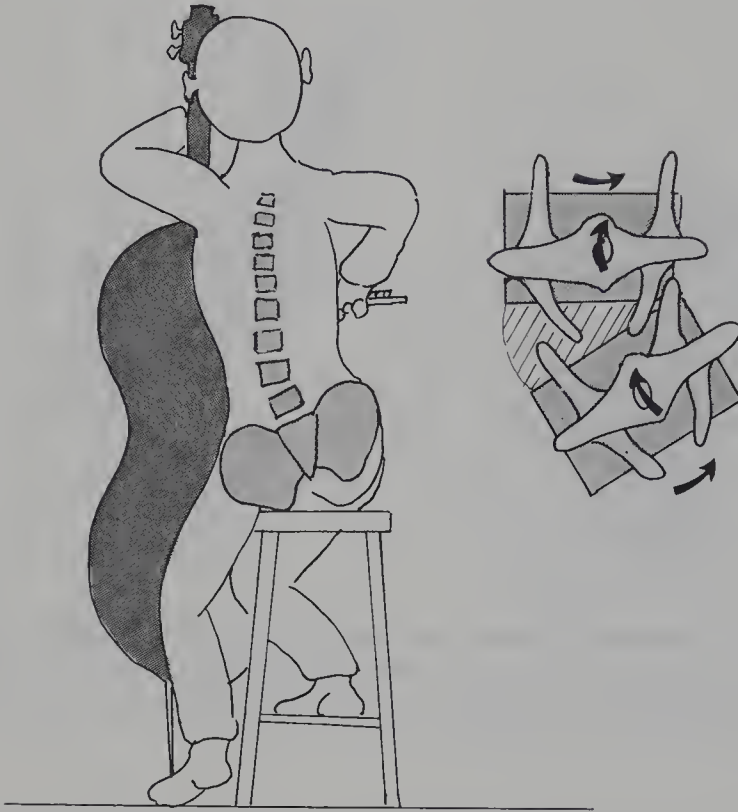


Figure 3-15. Pelvic angulation in a musician. Seated obliquely as required by functional position causes a pelvis obliquity with superincumbent scoliosis. The laterally curved lumbar spine (*small arrows*) functional unit causes an oblique deformity of the disk and approximates the facets of the concave side (*small side drawing*).

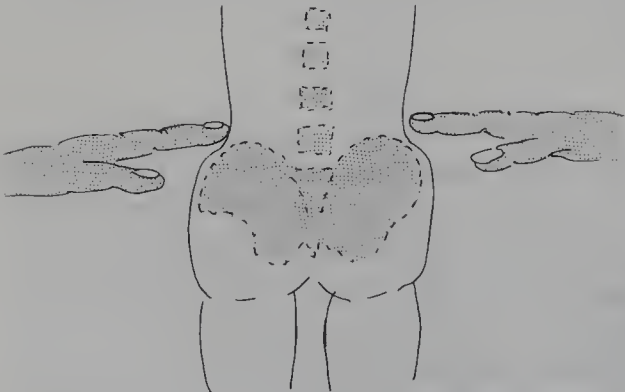


Figure 3-16. Clinical examination of pelvic level. To evaluate the level of the pelvis in the standing patient the hands of the examiner are placed on the brims of both pelvic rims and visually sighted.

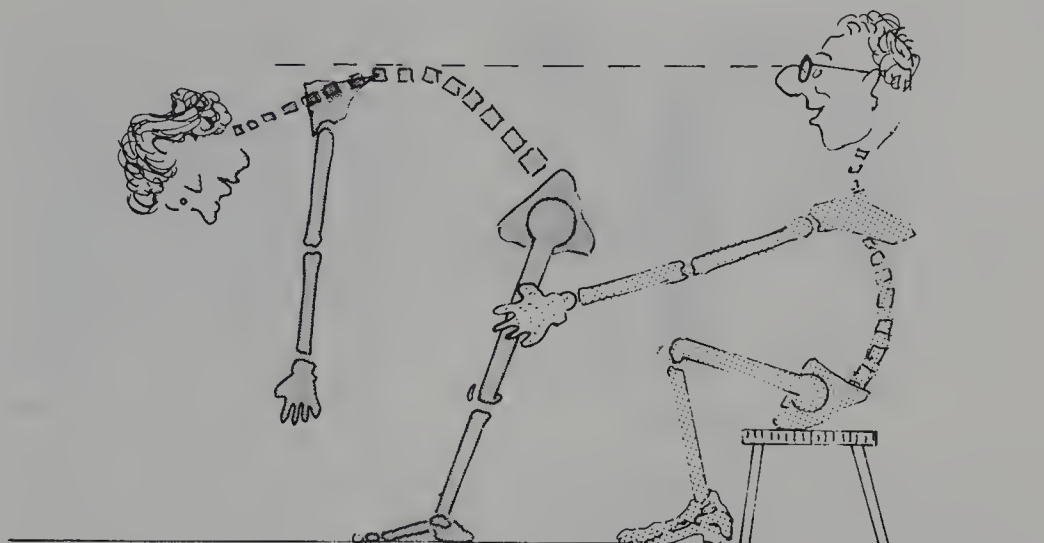


Figure 3-17. Clinical examination of scoliosis. To evaluate the degree of scoliosis and any rotation the seated examiner sights the flexed spine on the standing patient and determines the site of curvature and the degree of rotation.

## Lumbar Flexion

Forward flexion, which reverses the lumbar lordosis, has been tested and reported as a major aspect of the examination of the low back patient. ROM is documented in all medical reports. Its significance as well as its objective measurement remains limited except in patients with low back *and* leg pain in which disk pathology is contemplated.

Lumbar flexion is reduced in acute low back pain as a protective mechanism but is *not* reduced in patients with chronic low back pain.<sup>38-40</sup> It has validity in cases of spinal infection, tumor, herniated lumbar disk central and lateral with root irritation, but in benign chronic low back pain it has limited value and is difficult to accurately and objectively measure.

The limitation of the hamstring muscle elongation also influences lumbar flexion (Figs. 3-18 and 3-19). The influence of the hamstrings on lumbar flexion can be eliminated if trunk flexion is performed and observed in the seated position (Fig. 3-20).

Therapists have known for a long time that warm-up exercises preceding measurement of flexibility increases the range of motion tested.<sup>35-37</sup> Therefore, testing without warm-up preceding the test leads to variable results, making the objectivity of the test questionable.

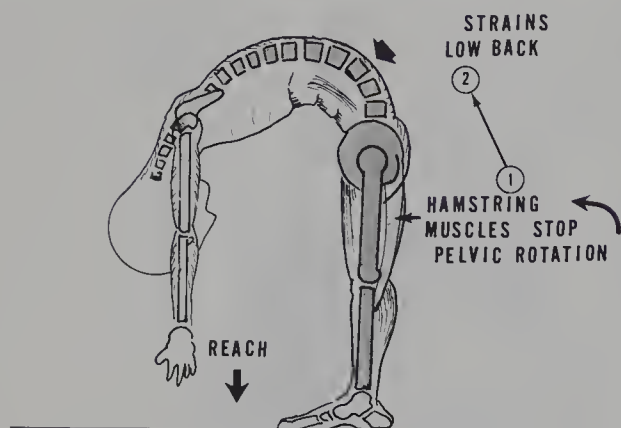


Figure 3-18. Standing test for degree of lumbar flexion and hamstring tightness. To evaluate the degree of lumbar flexion and where a loss of flexion occurs as well as determining the degree of hamstring tightness the patient is viewed from the side. The patient with legs extended flexes fully in an attempt to touch the floor ahead of the feet. The patient is asked to

flex fully but slowly to avoid low back strain. Pain may be produced in doing the test, which also indicates structural problems. In case of a sciatic neuritis this test causes pain radiation down the posterior aspect of the afflicted nerve root.

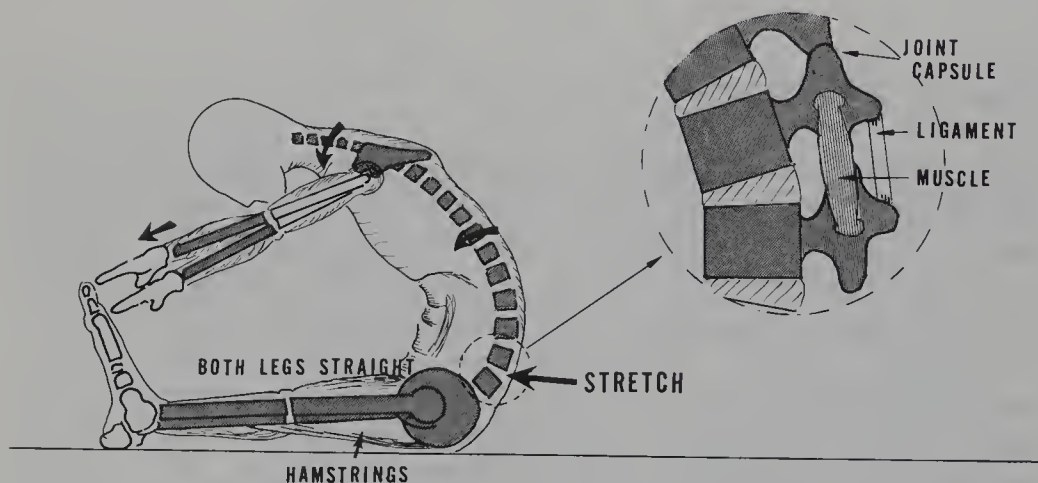
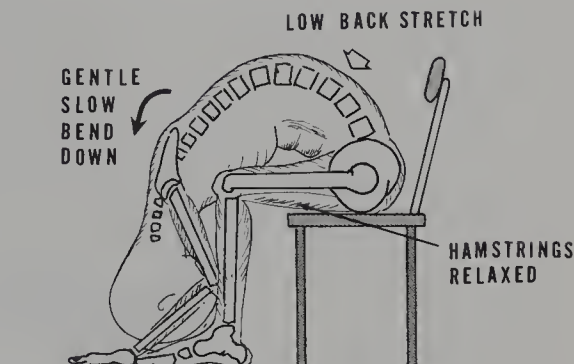


Figure 3-19. Supine test for degree of lumbar flexion and hamstring tightness. The same test as depicted in Figure 3-18 to evaluate the degree of lumbar flexion and where a loss of flexion occurs as well as determining the degree of hamstring tightness. The patient is viewed from the side. The supine patient with legs extended flexes fully in an attempt to touch the feet. The patient is asked to flex fully but slowly to avoid low back strain of the joint capsule, posterior ligaments, and erector spinae muscles (*circle*). Pain may be produced in doing the test, which also indicates structural problems. In case of a sciatic neuritis this test causes pain radiation down the posterior aspect of the afflicted nerve root.



Figure 3–20. Testing low back flexibility without hamstring involvement. Having the patient seated the hamstring muscles are totally relaxed. Having the patient flex forward demonstrates the flexibility of the lumbosacral spine.



Limited trunk flexion in discogenic disease is another premise and will be discussed under low back and leg pain. Its limitation in chronic and benign conditions, as stated, is neither consistent nor objective.

## Lateral and Rotational Flexibility

Both lateral flexibility and rotational flexibility are tested (Figs. 3–21 to 3–24) as being objective signs and measured by isokinetic machines. Their significance in the production of pain, however, remains unclear. They are highly influenced by pain and fear, and as behavioral signs thus are subjective signs.

Lumbar flexibility has been considered a valid objective test of impairment yet is basically a test of disability and highly subjective as it is influenced by disuse, fear, and pain.<sup>41–44</sup>

## Hamstring Flexibility

When trunk flexion is considered to be impaired by inflexible hamstring muscles (Fig. 3–25), this must be tested and accurately evaluated. If the limited straight leg raising is considered exclusively muscular and fascial and not from nerve root tension, pain will be considered muscular and there will be negative dural signs.<sup>27</sup> What this means is that the pain will be unilateral and not significantly aggravated by ankle dorsiflexion and simultaneous nuchal flexion. This will be discussed more thoroughly in the Neurological Examination section of this chapter.

## SIDE BENDING 1

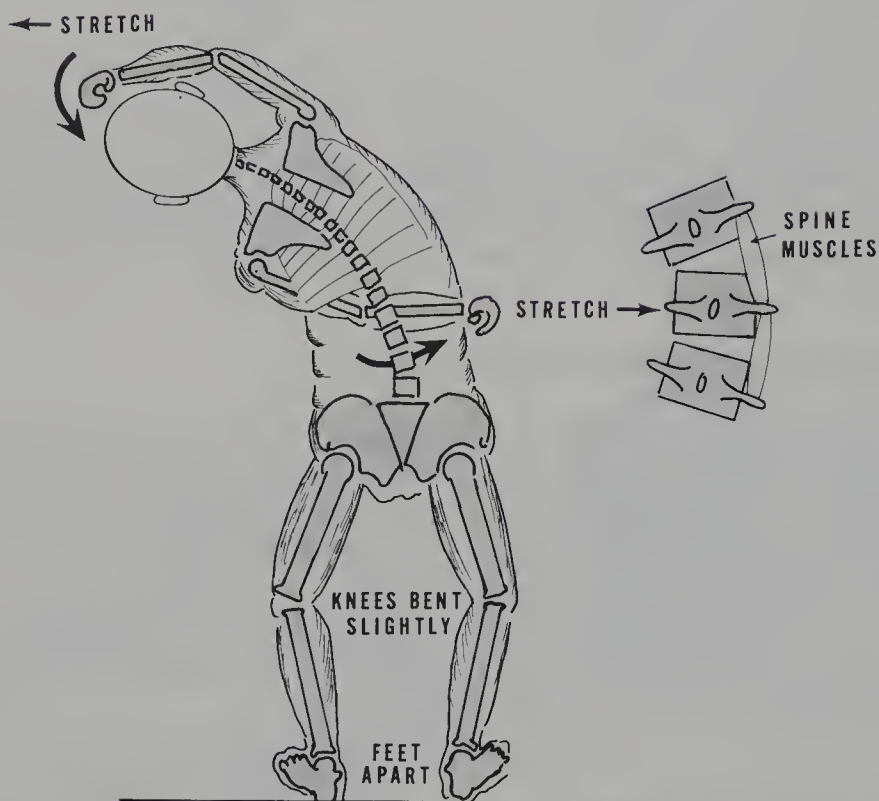


Figure 3-21. Testing lateral trunk flexibility. To test the flexibility of the lateral trunk soft tissues the patient, standing with feet slightly apart and the knees slightly bent, is asked to bend as far as possible to one side then the other. The small drawing depicts the tissues being tested. Bringing the arms overhead to either side also tests the flexibility of the latissimus dorsi muscle groups.

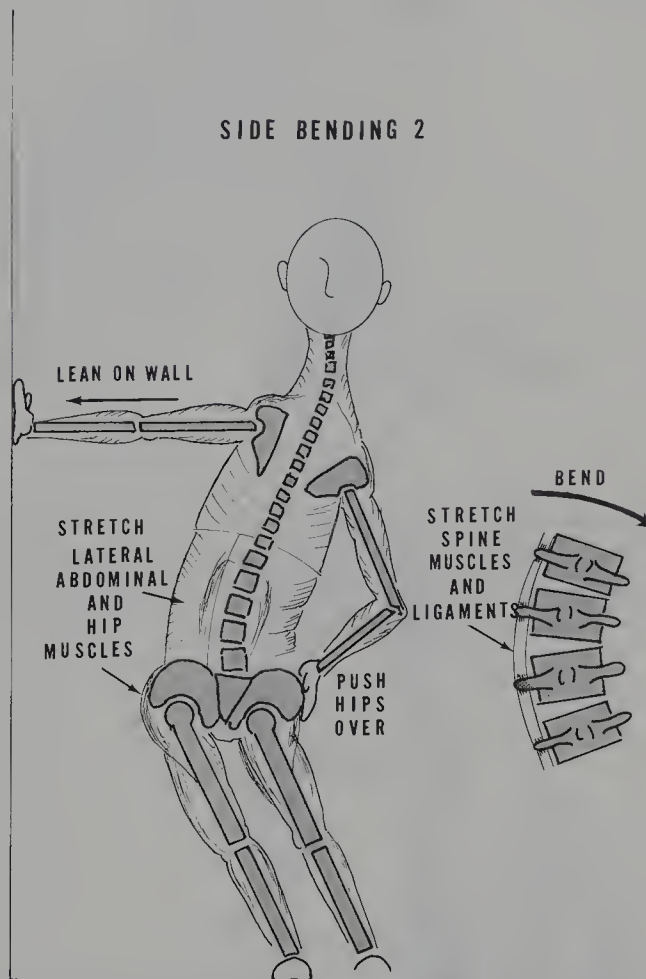


Figure 3-22. Another test of lateral trunk flexibility. To test the flexibility of the lateral trunk soft tissues the patient, with feet together, is asked to lean against a wall. With the contralateral hand the pelvis is pushed toward the wall. This maneuver tests the flexibility of the lateral abdominal and hip muscles as well as the paraspinous muscle and ligaments (side figure).

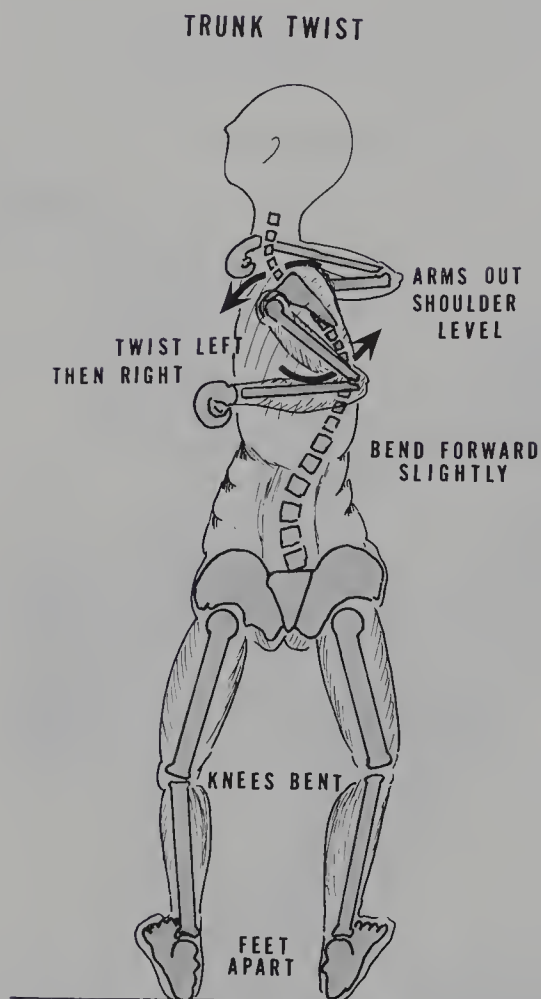
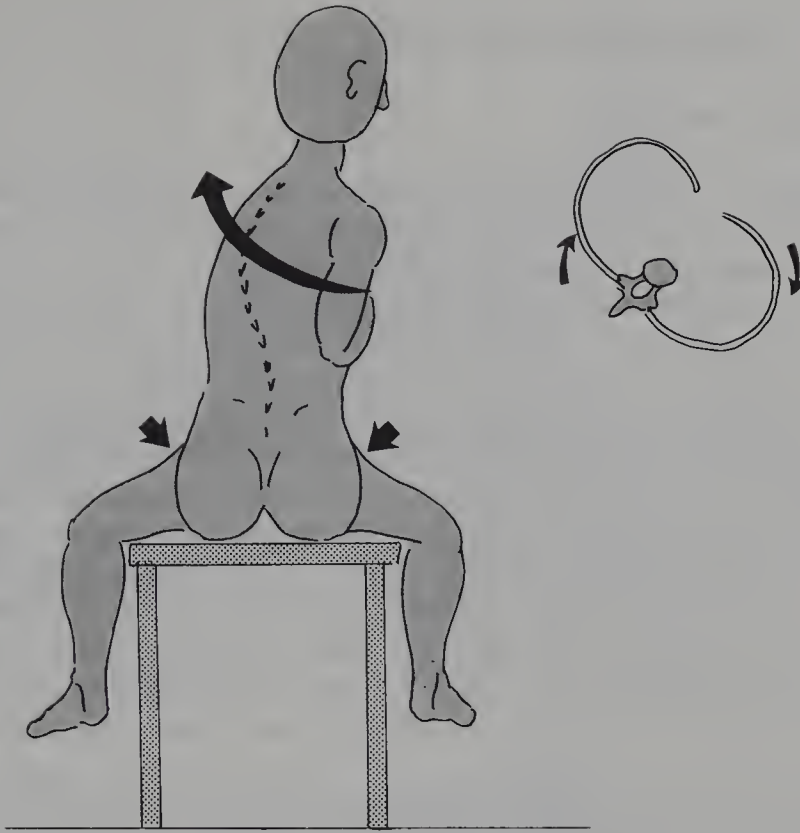
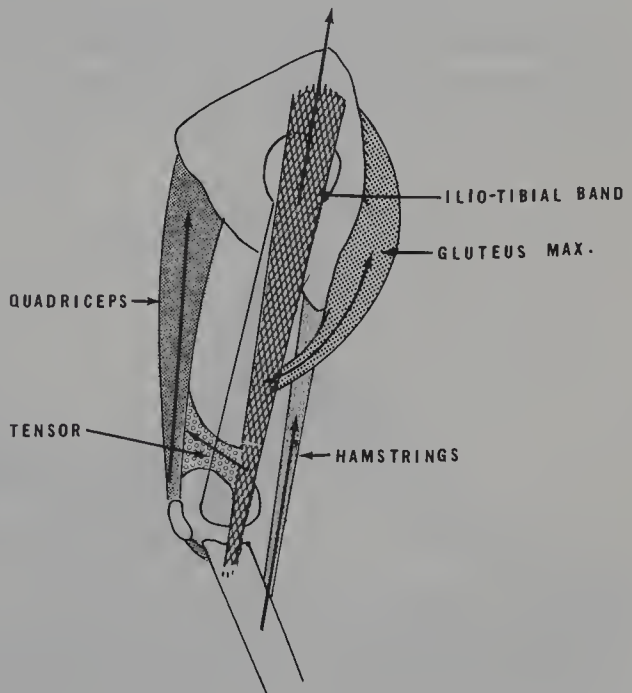


Figure 3-23. Testing trunk rotational flexibility. To test the rotational flexibility of the trunk the patient stands with legs extended and knees slightly bent and feet slightly apart. The patient actively turns the upper trunk to the right then the left. The degree of rotation from left to right is estimated and compared.



**Figure 3-24.** Testing trunk rotational flexibility in seated position. To test the rotational flexibility of the trunk the patient sits saddled over the examining table, which fixes the trunk. The patient actively turns the upper trunk to the right then the left. The degree of rotation from left to right is estimated and compared.



**Figure 3-25.** Muscles acting on the pelvis. In evaluating trunk flexibility the muscles acting on the pelvis are also involved. Their flexibility should also be individually evaluated. These tissues are the iliotibial band, the gluteus maximus, the quadriceps muscles group, the tensors, and the hamstrings.



## Strength Testing

Strength testing of the trunk muscles as an objective test for the cause of low back pain symptoms has also been advocated and has led to instrumentation for documenting this weakness.

The method of testing abdominal strength is the sit-up, which can be done properly or improperly and thus casts question on the validity of the test. Done with the knees and hips flexed and in stages (Fig. 3-26), the test can be performed without causing pain and be valid.<sup>45</sup>

Abdominal strength can best be tested from the shortened (fully flexed) position and gradual descent (Fig. 3-27). It can be tested with the legs extended (Fig. 3-28). In a physically unfit person this sit-up can cause painful lordotic extension (Fig. 3-29). Because of the resultant low back pain, it cannot be considered a valid test of strength.

Testing abdominal strength by bilateral straight leg raising (Fig. 3-30) is also feasible *if* the person has strong abdominal muscles, but in a deconditioned person low back pain can result, making the test invalid.

Extensor muscle strength testing can be done by manual resistance of lifting variable weights. In this test the way the patient lifts can also be evaluated (Fig. 3-31).

Objective measurement of trunk strength is given a full chapter (Chapter 7) and will not be repeated here. It must be remembered that most strength tests are invalidated by pain, pain-fear avoidance, deconditioning, and failure to make maximum effort. An objective test is therefore not gained. It must also be noted that muscle strength tested in the usual manner does not indicate this as being or having been the mechanism of chronic low back pain.

SITUP : STAGES

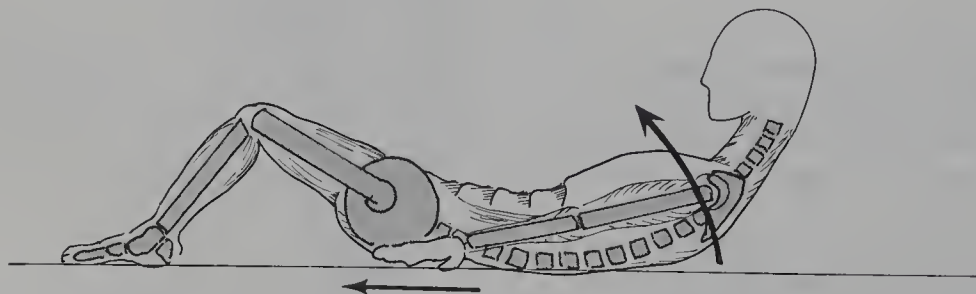
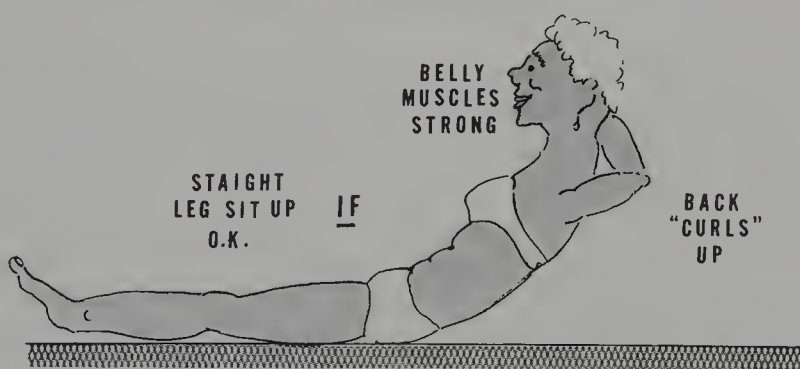


Figure 3-26. Stages of trunk flexor strength testing. The trunk flexors, or the abdominal muscles, are best tested in stages of sitting up from the supine position. These stages are also used in exercises for trunk strengthening. The first stage is the elevation (flexing) of the head and neck, which causes a coordinated contraction of the abdominal muscles.



**Figure 3-27.** Sit back abdominal strength testing. With patient seated and the feet possibly held down, the patient slowly sits back and holds the position at various degrees. This test can be done with arms extended forward if poorly conditioned or arms behind the head if reasonably strong.



**Figure 3-28.** Abdominal strength testing. Sit up with legs extended. From the supine position with legs extended, and feet possibly held down, the patient sits up. This test is not advised if abdominal muscles are extremely weak as it causes a lumbar lordosis that may be painful.



**Figure 3-29.** Poorly conditioned patient doing a painful sit up. From the supine position with legs extended, and feet possibly held down, the patient sits up or sits back, which causes low back pain from hyperextension if the abdominal muscles are extremely weak.

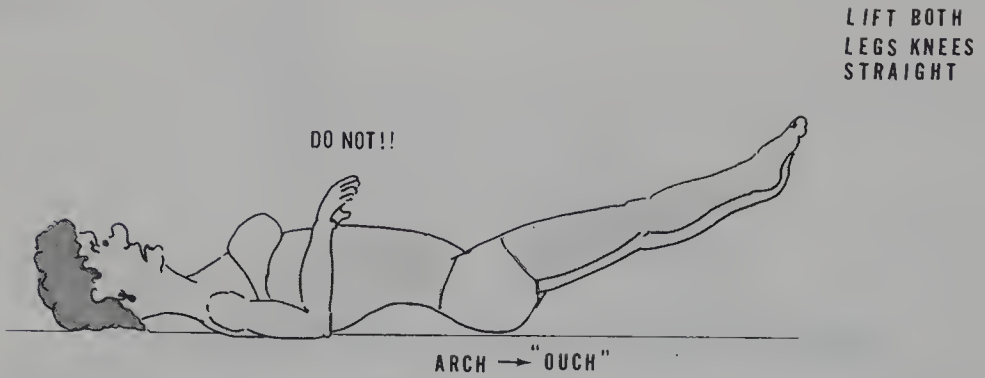


Figure 3-30. Abdominal strength testing with bilateral leg raising. If the patient has extremely weak abdominal muscles this test usually causes a painful low back extension, otherwise it can be used to test rectus abdominal muscle strength and endurance.

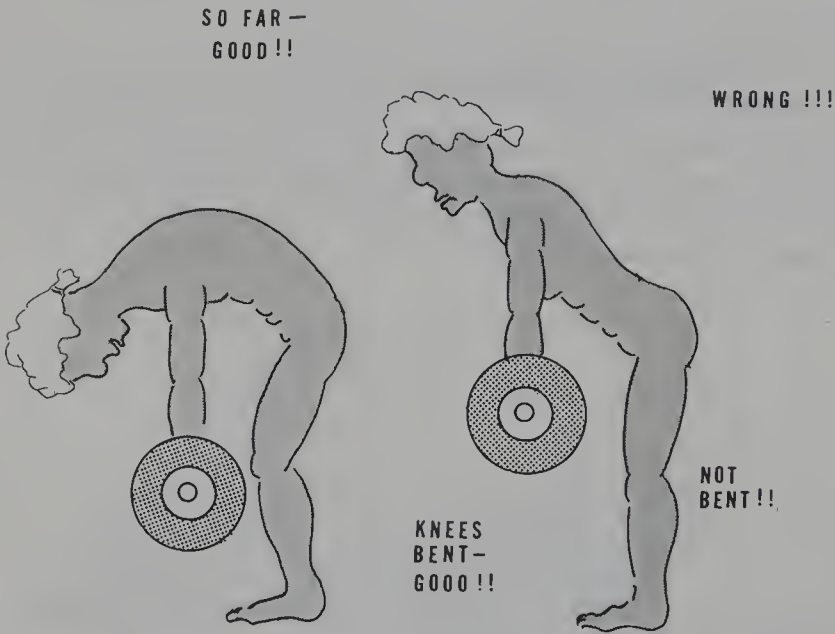


Figure 3-31. Extensor muscle strength testing. There are many machines designed to test trunk extensor strength (see Chapter 7). A simple test is to have the patient lift a measured weight. This test should conform to proper physiologic mechanism thus done with the legs and back slightly flexed to avoid extension during the lift.

## Lumbar Extension

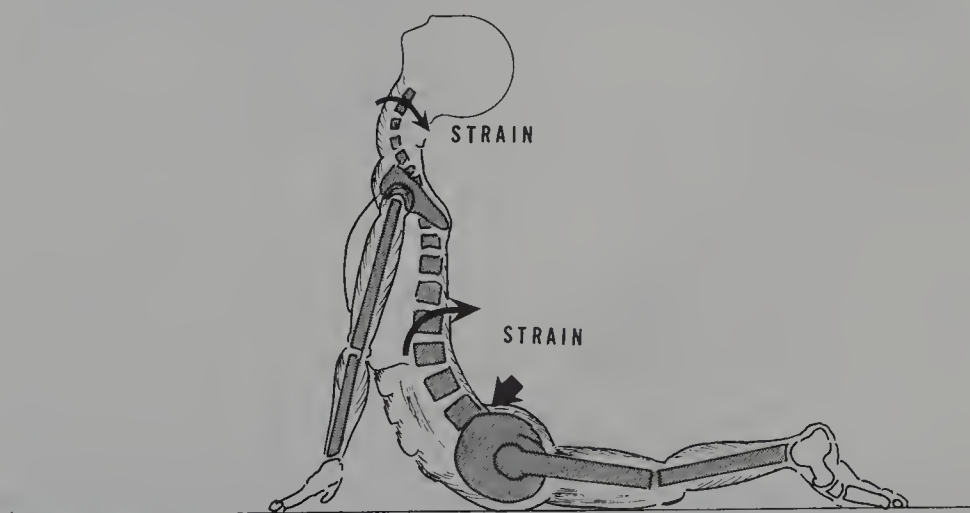
Lumbar extension has also been elicited to determine its causation of low back pain (Fig. 3-32). This test again demands exclamation of pain by the patient and thus is not wholly objective.

One of the mechanisms of pain causation on lumbar extension has been postulated to be foraminal closure, entrapping the nerve; impingement of the facets; or even a posterior disk bulge (Fig. 3-33). The newer concepts of McKenzie<sup>32</sup> imply that passive hyperextension can cause the internally herniated disk to migrate anteriorly (Fig. 3-34) and thus relieve a symptomatic bulge.

All these tests considered as objective still require patient effort, compliance, and performance, often in spite of pain. They are therefore subjective and need careful evaluation.

## Objective Neurological Examination

An objective test, if performed accurately, is the SLR test, otherwise termed the Lasègue test.<sup>46,47</sup> The objective of the SLR test is to determine whether the resultant pain elicited behind the thigh is muscular or nerve dura irritation. If it is dura irritation the sciatic pain can be produced by the normal leg raising test of Fajersztajn<sup>48</sup>, which causes the dura to move distally and laterally. Essentially the SLR test is merely extending the hip, with the lower leg extended, and eliciting pain and determining at which angle of flexion the pain occurs.



**Figure 3-32.** Confirming low back pain caused by extension. As claimed by the patient's history that hyperextension causes low back pain this can be confirmed by having the prone patient extend and hyperextend by active extension using the arms.

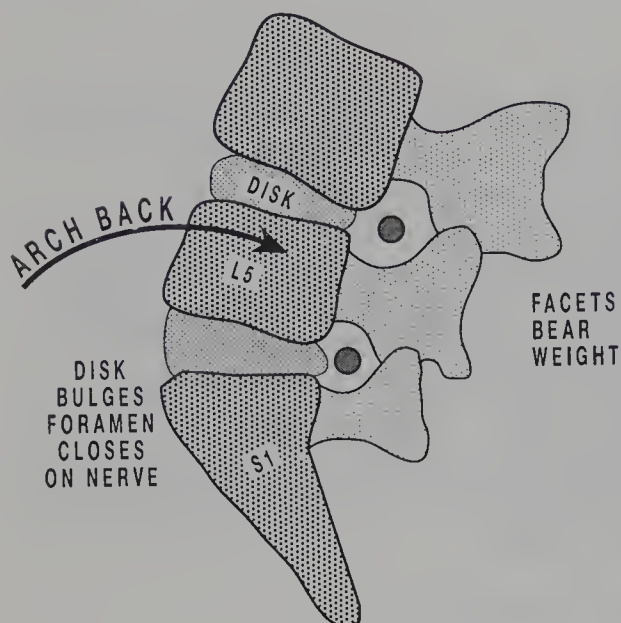


Figure 3-33. Lumbosacral hyperextension causing root pain: the postulated mechanism. As the lumbosacral spine extends (*top arrow*) the intervertebral foramen narrows and the disk protrudes, causing pressure on the nerve root. Pain can occur from weight bearing on the facets in this position.

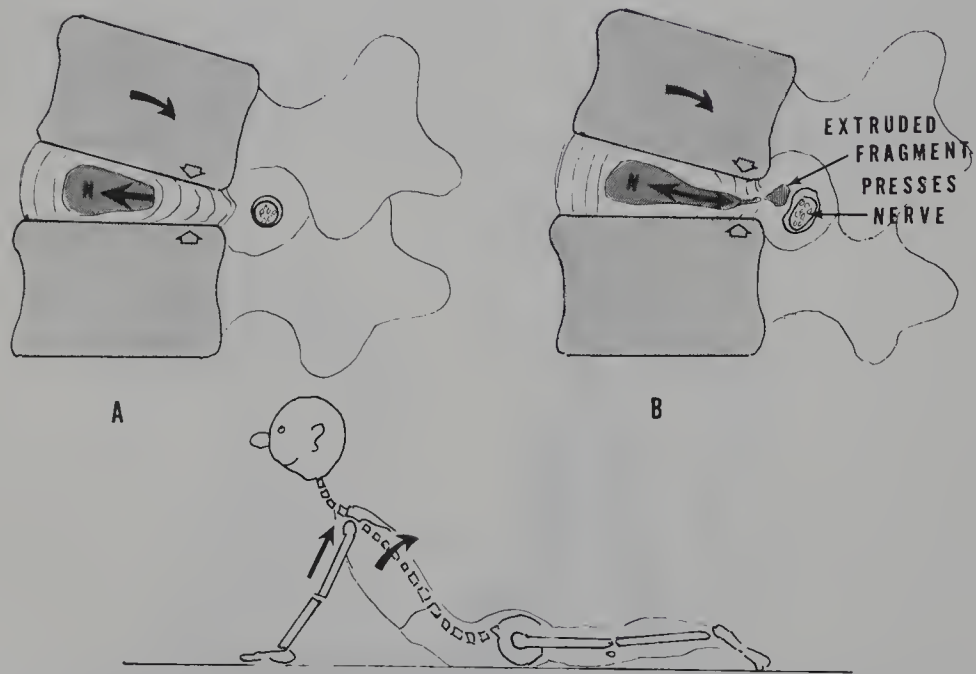


Figure 3-34. Lumbosacral hyperextension causing root pain from intervertebral disk protrusion. As the lumbosacral spine extends (lower figure) the nucleus (N) of the disk migrates anteriorly (A). If the disk is extruded, this position (B) encroaches on the nerve root and forces the fragment to compress the root.



The hamstring muscles, as has been discussed previously, elongate to their physiological flexibility as the extended leg is flexed. Resultant pain occurs as the myofascial component of the hamstring muscle group is shortened. The pain usually is identical in both legs at the same level and is identified as muscular in nature.

If pain occurs from performing the straight leg procedure, it must be determined if the resultant pain is from sciatic nerve stretch. In other words, whether there is a peripheral neuropathy. There presents a confusion in terminology clearly analyzed by van Akkerveeken,<sup>49</sup> differentiating the term *radicular pain* from *sciatica*. The former should be used for patients suffering from pain resulting from pathology of the spinal nerve root, whereas the latter is a neuralgia of the sciatic nerve trunk. This is not just a matter of semantics but a significant difference.

The term *sciatica* was derived from the Latin *ischialgia*, meaning “pain” (*algia*) and “buttocks” (*ischia*). *Sciatica* therefore meant pain in the lower buttocks and upper part of the posterior thigh. Hippocrates called this type of pain *hip pain*.<sup>50</sup> It was not until 1770 that Cotugno differentiated pain from the hip with pain from the sciatic nerve.<sup>51</sup> He also differentiated pain down the back of the thigh (termed *ischias nervosa postica*) with pain down the front of the thigh (termed *ischia nervosa antica*, or femoral radiating pain).

*True sciatica* was the term applied to neuritis of the sciatic nerve originally occurring from untreated diabetes and severe alcoholism. It was Wertheim-Salomonson who differentiated nerve root neuralgia from nerve trunk neuritis.<sup>52</sup>

As *neuritis* (inflammation) of the nerve may not be present, that term should be reserved where inflammation is ascertained. Radicular pain is localized in the area of dermatomes, whereas *sciatica* is in the distribution of the sciatic nerve trunk.

Pain resulting from direct stimulation of nerve root fibers usually occurs from neural injury and should probably be termed *deafferentation pain*.<sup>53,54</sup> This is veritably radicular pain, implying pathological processes inside the spinal nerve root.

Mechanical pressure on normal nerves affects the large diameter fibers first with resultant impairment of sensation and power, whereas the smaller fibers transmit nociception.<sup>55</sup> Further pressure on normal nerves causes paresthesia not pain.<sup>56</sup> Pain occurs from pressure on a previously inflamed nerve.<sup>57</sup> Pressure on a nerve as the cause of radicular pain should be preceded by loss of sensation and motor power.

Verbiest postulated that pain from compression of a nerve root occurred from irritation of the *nervi nervorum* of the root sheath when he eliminated the pain from an inflamed nerve root by placing a drop of cocaine on the sheath.<sup>58,59</sup>

Pain down the distribution of the nerve root also occurred from irritation of the ventral roots,<sup>60</sup> which had been considered strictly motor and not sensory. Frykholm presented this fact by electrical stimulation of motor roots causing pain.<sup>61</sup> Interestingly, blocking the posterior root abolished this sensitivity.

Some concluded that the site of nerve root compression is from herniation of nuclear disk material, but the hypersensitivity of the nerve root also was found to occur from the remaining annulus that was not protruding and also from the posterior longitudinal ligament covering the remaining disk.<sup>62</sup>

Painful straight leg raising is thus considered a nerve and not a muscle stretch phenomenon and is termed a *positive SLR*. The basis for nerve root irritation of a nerve root nerve, causing a positive test, may be a disk herniation (Chapter 5), facet arthritic changes in the foramen (Chapter 6), direct trauma to the sciatic nerve, or a tumor along its course from the cord. This is true radiculitis, whereas true sciatica results from a nerve and neuritis results from other sources.

SLR tests are performed in the following ways.

1. With the leg fully extended at the knee, it is passively flexed at the hip joint.
2. With the person seated, and thus the hip flexed to 90°, the lower leg can be passively extended.

Both of these tests only test the muscular flexibility of the posterior thigh muscles. The sciatic nerve is also extended but is not sensitive, thus causing no pain unless inflamed under tension. If the nerve is inflamed and under tension, the SLR is limited, with pain occurring early in straight leg raising and usually only in one leg.

To determine that the limited straight leg raising is from irritable nerve stretching and not merely muscle, dural signs are then tested. As has been stated, the dura of the nerve root(s) is highly innervated and thus is the tissue site of the nerve that elicits pain when stretched.

These dural signs are elicited by extending (stretching) the dura of the nerve. This is done by flexing the neck or dorsiflexing the foot-ankle after accomplishing full passive straight leg raising (Figs. 3-35 and 3-36). It is obvious that mere forward flexion of the trunk with the legs extended also causes a straight leg raising motion, which when followed by nuchal flexion imposes a dural test.

A confirmatory dural test is the Fajersztain test,<sup>48</sup> known as the *well leg raising test* or *crossed nerve stretch*. If positive, the sciatic pain radiation occurs down the afflicted leg from raising the opposite, normal leg. The basis for this is that the inflamed dura of the afflicted leg is drawn over as the well leg is extended.

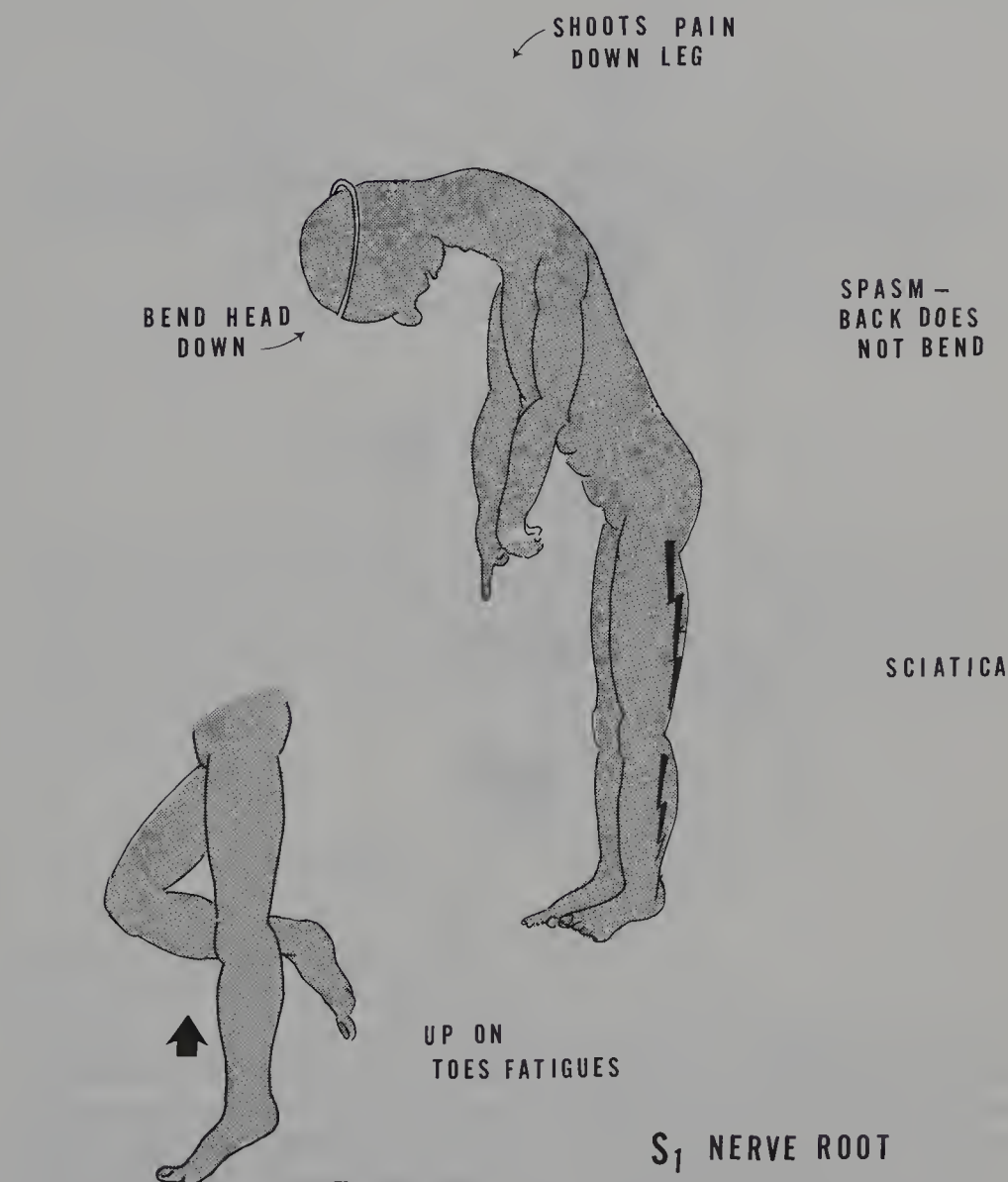


Figure 3-35. Straight leg raising test (SLR) from standing position: the dural sign. In the presence of a herniated lumbar disk as the patient flexes forward with legs extended the low back does not flex and the straight leg is essentially flexed (SLR). This alone may cause sciatic neuritic pain. The dural sign is then elicited by flexing the neck and head. Suspecting an S<sub>1</sub> root involvement the strength of the gastrocnemius and soleus muscle (lower drawing) is tested by having the patient arise repeatedly on the toes. Fatigue or weakness confirms S<sub>1</sub> involvement.

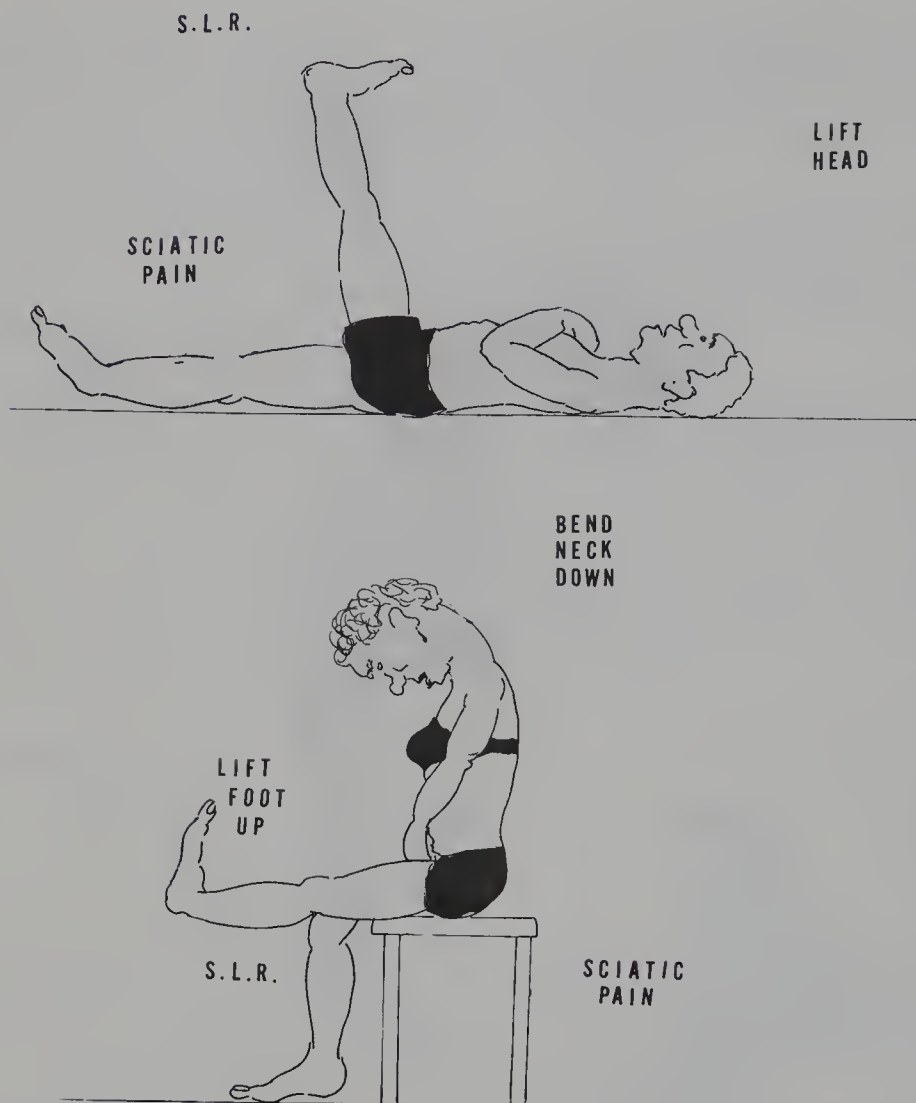
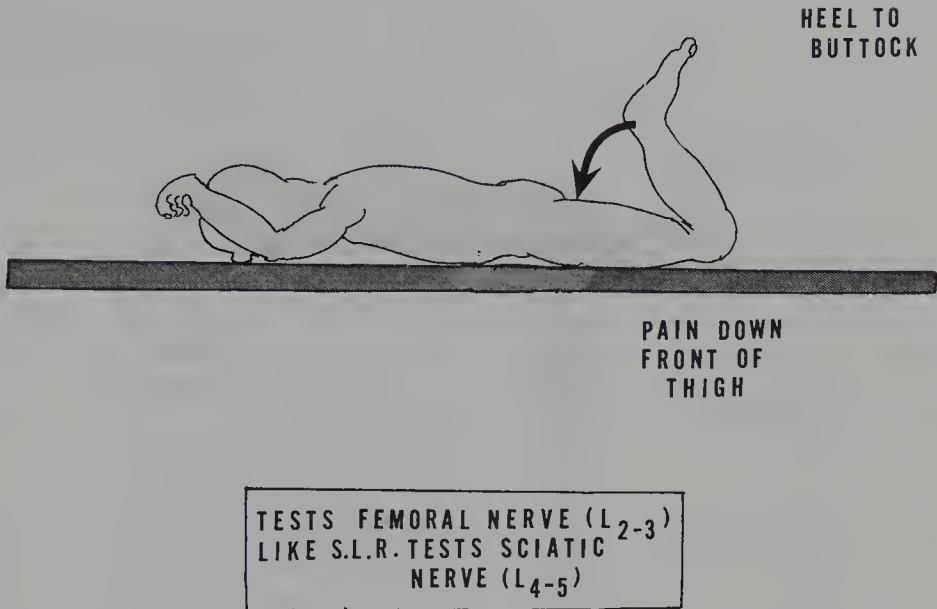


Figure 3-36. Straight leg raising test (SLR) in the supine and in the sitting position. The top drawing shows the method of eliciting nerve root pain from straight leg raising. The extended leg is slowly flexed at the hip and compared with the other leg. Flexing the head and neck elicits the dural sign as would dorsiflexing the foot after raising the leg (not illustrated). The bottom figure demonstrates the same test in the seated position. Here the dural test includes the ankle dorsiflexion as well as nuchal flexion.

Straight leg raising only tests the lower root segments (L4, L5, S1) as only these roots extend into the lower leg. If the neuritis, from whatever cause, involves upper lumbar segments (L2, L3) that form the femoral nerve, they are not stretched from SLR. This must be tested with the femoral stretch test (Fig. 3-37). This is performed with the patient prone, thus having the hip extended, and the knee flexed (the foot approaching the buttocks). This stretches the quadriceps as well as the femoral nerve, but there is no known dural component to the test.





**Figure 3–37.** Femoral stretch test. When the upper lumbar nerve roots are entrapped (L2, L3, and possibly L4), which descend the femoral nerve, the test is the prone femoral nerve test. With the patient prone the lower leg is flexed to approach the foot to the buttocks. Pain is felt down the front of the thigh. It must be differentiated from muscle (quadriceps) stretch pain and be compared with the other leg.

## Neurological Determination of Nerve Root Involvement

As all lower extremity muscles and sensory nerves emit from the lumbosacral spine, each must be individually tested to determine its being entrapped, inflamed, or under tension and at which spinal level.

Clinical dermatome and myotome levels are depicted (Fig. 3–38). All muscles are innervated by peripheral nerves that contain more than one nerve root (Table 3–1).

The dermatomal areas of the lower extremity that are subserved by nerve roots also have specific nerve roots for each dermatome (Table 3–2).

Dermatome areas can be mapped out by the examiner by rubbing the skin with a piece of cotton, the end of a pin, or a wheel. The patient literally draws the map as he or she indicates feeling the test object. Dermatomal mappings are in every neurological test (Fig. 3–39) and are usually those of Keegan and Garret<sup>63</sup> (Fig. 3–38), Bolk,<sup>64</sup> or Hansen and Schliack.<sup>65</sup>



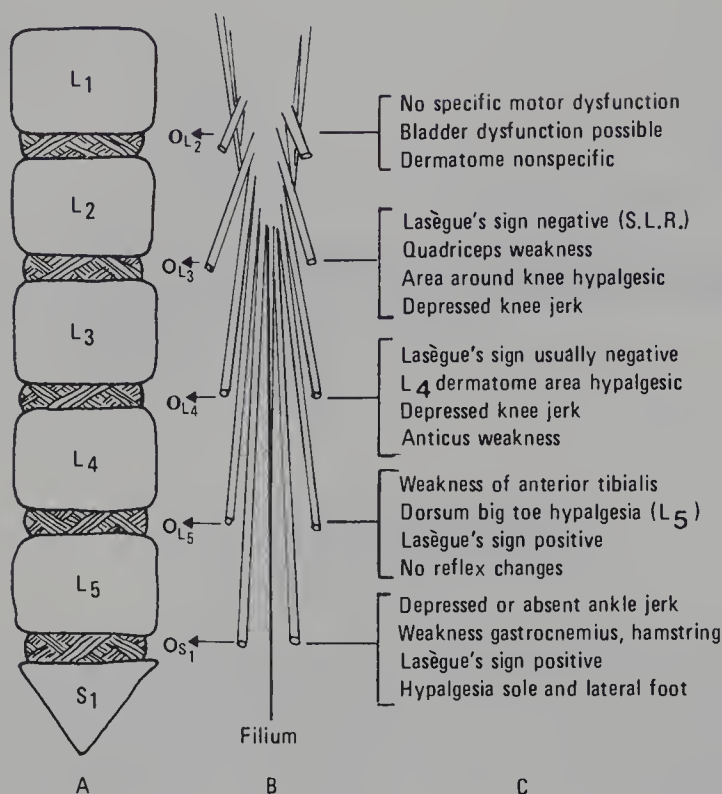


Figure 3-38. Clinical localization of dermatome-myotome level of disk herniation. Lateral view of the spine showing the nerve root relationship to the intervertebral disk (e.g., S1 nerve root at level of disk between vertebral L5 and first sacral vertebra S1) (A). Indication of the level at which the cord becomes the cauda equina (L1) (B). Below this level any nerve injury is equivalent to a peripheral nerve injury. The clinical manifestations of the various nerve roots as found on neurologic examination (C).

Variations in the dermatomal areas have been raised in which the L5 syndrome differed in patients from pain in the posterior thigh along the course of the sciatic nerve, some at the posterior popliteal area, lateral aspect of the calf and into the foot in the dorsolateral aspect of the big toe. Those with S1 dermatomal radiation had pain in the posterior aspect of the thigh and calf and the lateral aspect of the foot. It is apparent from this thorough evaluation that the precise dermatomal area to designate a specific nerve root is inaccurate.<sup>49</sup>

The only conclusion derived was that the pain pattern of patients with disk herniation did not differ from the pain pattern caused by degenerative stenosis with entrapment. No patient with an S1 lesion had pain in the big toe but did have pain in the lateral aspect of the foot, whereas L5 lesions tended to be localized in the dorsum of the foot.

Table 3-1. RELATIONSHIP OF SPECIFIC ROOTS, MUSCLES, AND PERIPHERAL NERVES

<i>Root</i>	<i>Muscle</i>	<i>Peripheral Nerve</i>
L2	Sartorius (L2-3)	Femoral
	Pectineus (L2-3)	Obturator
	Adductor longus (L2-3)	Obturator
L3	Quadriceps femoris (L2-3-4)	Femoral
L4	Quadriceps femoris (L2-3-4)	Femoral
	Tensor fascia lata (L4-5)	Superior gluteal
	Tibialis anterior (L4-5)	Peroneal
L5	Gluteus medius (L4-5 S1)	Superior gluteal
	Semimembranosus (L4-5 S1)	Sciatic
	Semitendinosus (L4-5 S1)	Sciatic
	Extensor hallucis longis (L4-5 S1)	Deep peroneal
S1	Gluteus maximus (L4-5 S1-2)	Inferior gluteal
	Biceps femoris—short head (L5 S1-2)	Sciatic
	Semitendinosus (L4-5 S1)	Sciatic
	Medial gastrocnemius (S1-2)	Tibial
	Soleus (S1-2)	Tibial
	Biceps femoris—long head (S1-2)	Sciatic
S2	Lateral gastrocnemius (S1-2)	Tibial
	Soleus (S1-2)	Tibial

Herlin disagreed in that he claimed that “the L5 root caused pain in the lateral part of the gluteus with radiation down the posterolateral aspect of the thigh, the lateral aspect of the calf, lateral malleolus, dorsum of the foot ‘and the big toe’.” He claimed that “S1 lesions were located more posteriorly into the heel and along the lateral edge of the foot ‘into the big toe’.”<sup>66</sup>

This raises the question as to whether radicular pain is the direct result of nerve root insult or arises from the sheath around the nerve root.<sup>67</sup> Smyth and Wright demonstrated that the extent of the pain pattern was directly related to the magnitude of the mechanical impulses, with greater pressure on the nerve causing more distal pain patterns.<sup>68</sup>

In summary pain along the course of the sciatic nerve in the posterior thigh and the lower leg is probably caused by truncal neuritis from trauma to the sciatic nerve or from the piriformis muscles, whereas nerve root radiation (disk herniation) is from de-efferentation of a root and has dermatomal localization.<sup>50</sup>

The dermatomal area has already been designated by the subjective claim of the patient during the history taking as to “where the pain is felt,” especially during the SLR.

Table 3-2. SCHEMATIC DERMATOME AND MYOTOME LEVEL OF NERVE  
ROOT IMPINGEMENT

Nerve Root	Intervertebral Space	Subjective Pain Radiation	Bladder and Bowel			Motor Dysfunction (Myotome)f		
			Sensory Area	Dysfunction*	SLR†	Ankle Jerk‡	Knee Jerk‡	
L3	L2-L3	Back to buttocks to posterior thigh to anterior knee region	Hypalgesia in knee region	+/-	Usually —	+	+	Quadriceps weakness
L4	L3-L4	Back to buttocks to posterior thigh to inner calf region	Hypalgesia inner aspect of lower leg	+/-	Usually — Maybe +	+	—	Quadriceps and possible anticus weakness
L5	L4-L5	Back to buttocks to dorsum of foot and big toe	Hypalgesia in dorsum foot and big toe	+/-	++ ++	+	+	Weakness of anterior tibialis, big toe extensor, gluteus medius
S1	L5-S1	Back to buttocks to sole of foot and heel	Hypalgesia in heel or lateral foot	+/-	+++	—	+	Weakness of gastroc- nemius, hamstring, gluteus maximus

\*Bladder and bowel dysfunction can occur at any level.  
†Related to extent of nerve root movement of each level.  
‡Ankle jerk is absent only at L5-S1; knee jerk at L3-L4.  
§Only the more obvious and functional muscles are listed.

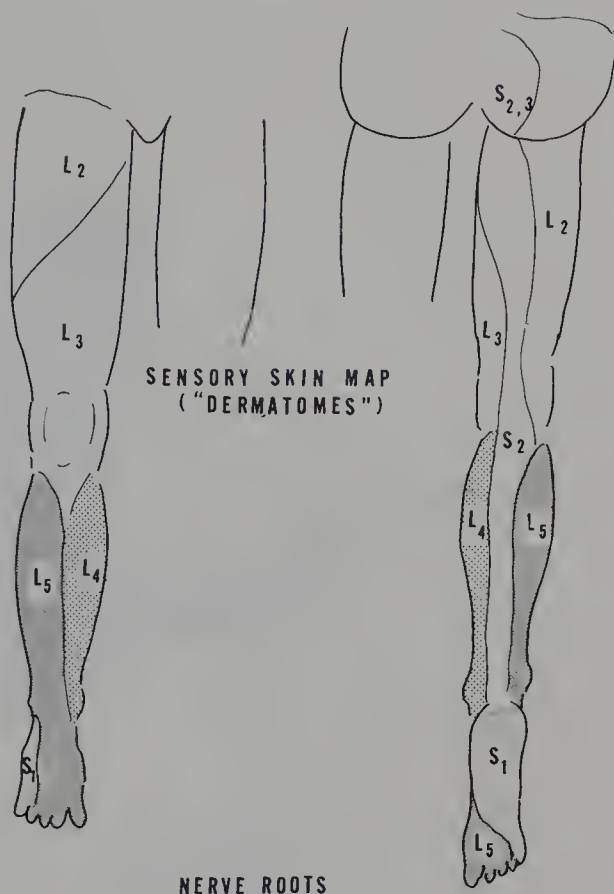


Figure 3-39. Dermatomal areas of the lower extremity. This is the classic dermatomal mapping of Keegan.

The myotome designation is performed by the patient actively initiating the precise muscle group and the nerve supply to that muscle group designates the dermatomes involved. Here again the objectivity of this test is colored by the willingness of the patient to fully cooperate.

The S1 nerve root innervates the gluteus maximus and the gastrocnemius group. The former is tested by the patient in the supine position, with the tested leg being flexed and the foot prone on the table elevating the buttocks several times. This tests the hip extensors, which are primarily the glutei (S1-2 and L4-5). This is compared to the other side, and endurance is tested by repeating the effort several times on both legs.

The gastrocnemius-soleus (S1-2) is tested by asking the patient to rise up on the toes of each leg separately and comparing the two. Endurance here is also tested by having repeated rising. Merely walking on one's toes is not satisfactory (Fig. 3-40).

Tenderness of the myoneural junctions has been postulated as having localizing characteristics (Fig. 3-41) but demands a careful search by the examiner's probing finger.<sup>69</sup>

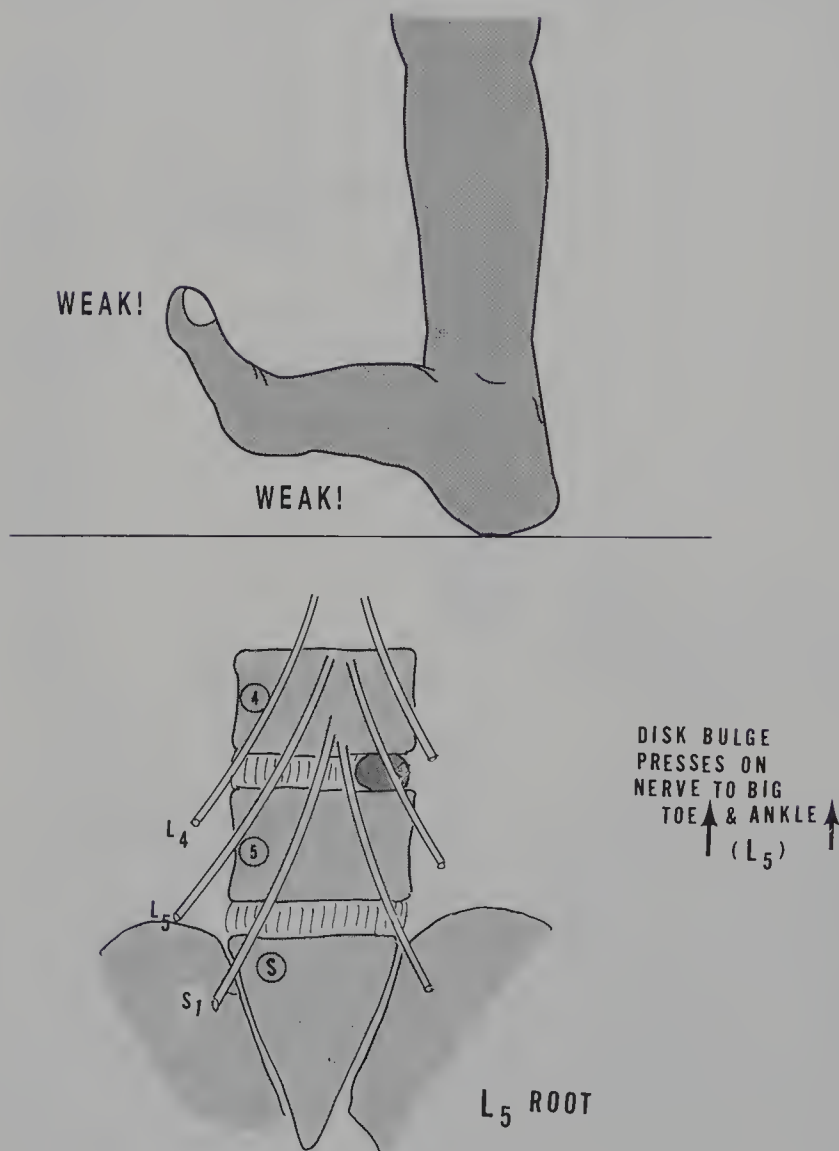
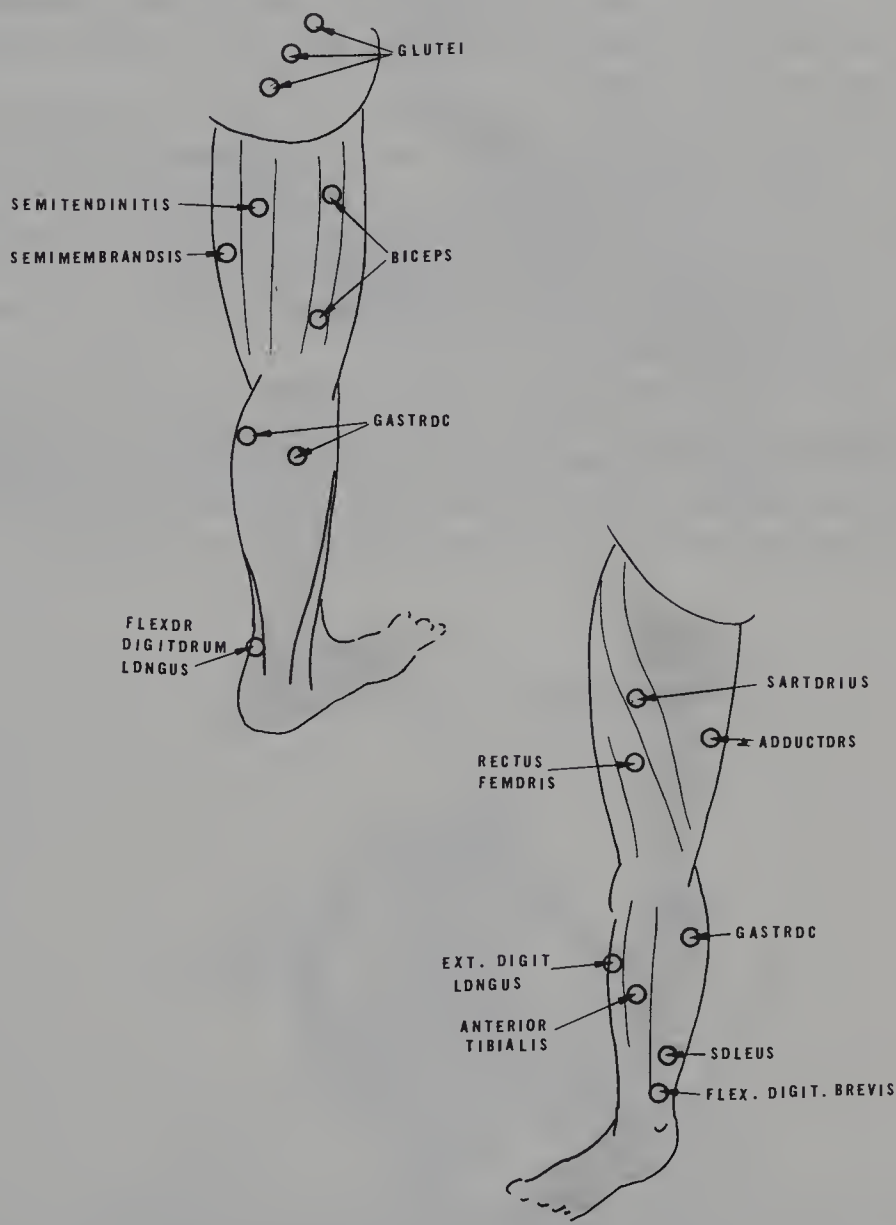


Figure 3-40. Testing myotome of L5 root. Testing the myotome of L5 indicates weakness or fatigue of the extensor hallucis longus (big toe extensor). This usually indicates an L5 lesion although some clinicians also implicate the S1 root. Fatigue as compared with the other side, is diagnostic.





**Figure 3-41.** Tender myoneural junction points. Deep pressure tenderness at the sites of myoneural junctions have been advocated to localize more central nerve root lesions. The innervations of the palpated muscles must be known and are often multiple innervations not merely one root.

## MISCELLANEOUS OBJECTIVE TESTS

Another test considered to be objective in the evaluation of the patient with chronic low back pain is tightened hamstring muscles (Fig. 3-42), causing asymmetry in total trunk flexion or even limitation of trunk flexion, preventing pelvis rotation and thus causing excessive strain on the lumbar spine.

Other tests frequently recorded are (1) passive knee flexion (Fig. 3-43), (2) passive hip flexion (Fig. 3-44), and (3) passive hip extension (Fig. 3-45). Hip abductor testing merely means seeing how far apart the legs can be abducted, but this is difficult to estimate as a variance of normal as these standards have not been established.

Hip extensor strength is also tested (Fig. 3-46), but this also is difficult to standardize.

Most of these miscellaneous tests attempt to specify if there is limitation or weakness of tissues about the hip and knee. Their role in causation of chronic low back pain is not clear; neither is their deviation from normal defined. The causation of pain from any of these tests is also subjective, thus making the tests themselves not objective.<sup>70</sup>

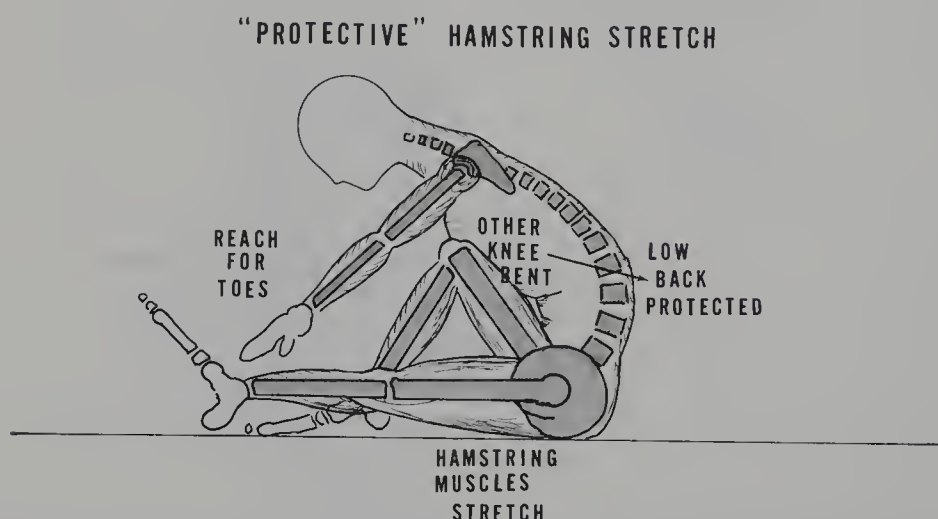


Figure 3-42. Unilateral hamstring stretch test. With the patient supine and seated upright the leg to be tested remains extended and the other flexed. It is the extended leg that is tested for hamstring limitation. Pain determines limitation as compared with the other side or to the expected normal. The nuchal flexion and ankle dorsiflexion is performed only to determine that limited flexibility is from the nerve roots and not merely the muscle. The other flexed leg protects the low back from being overstretched hence the term *protective hamstring stretch*. This is observed when this activity becomes a stretch exercise.

### KNEES TO CHEST

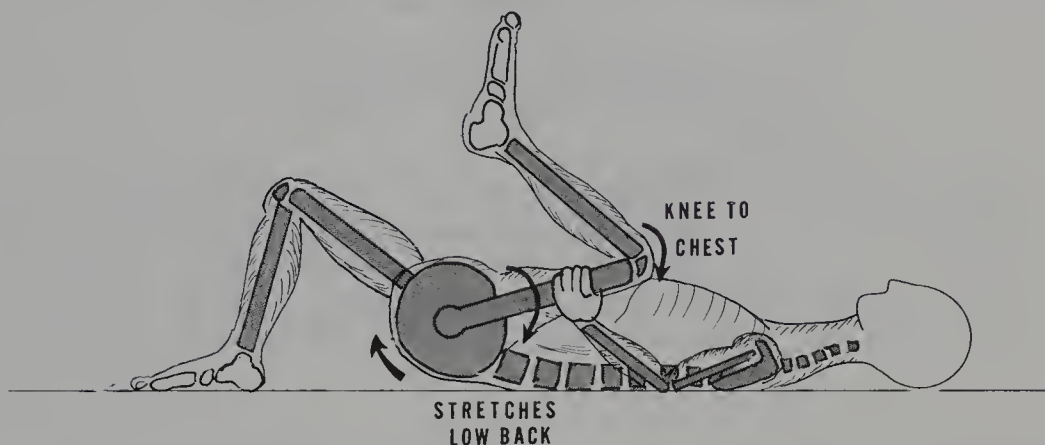


Figure 3-43. Passive knee flexion. This tests the range of motion of the hip and the knee and can become an exercise.

### HIP STRETCH

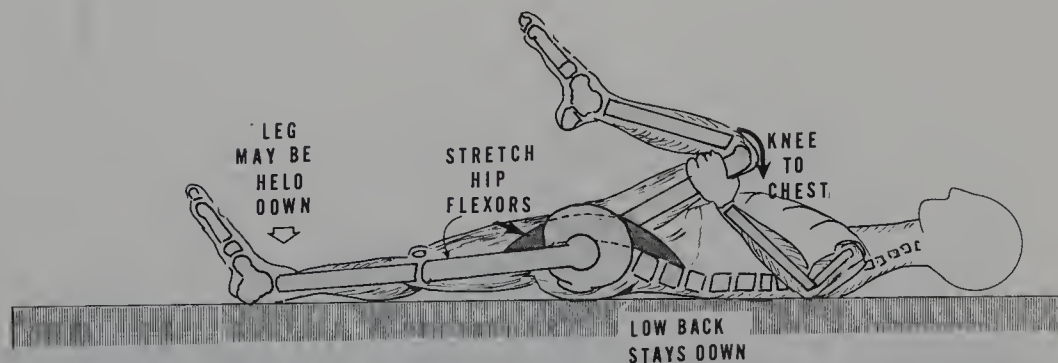


Figure 3-44. Hip flexor stretch test. This tests the range of motion of the hip flexors for flexibility. As the patient actively flexes the opposite leg to the chest the remaining extended leg reveals any hip flexion contracture. It can become an exercise to stretch the flexors.

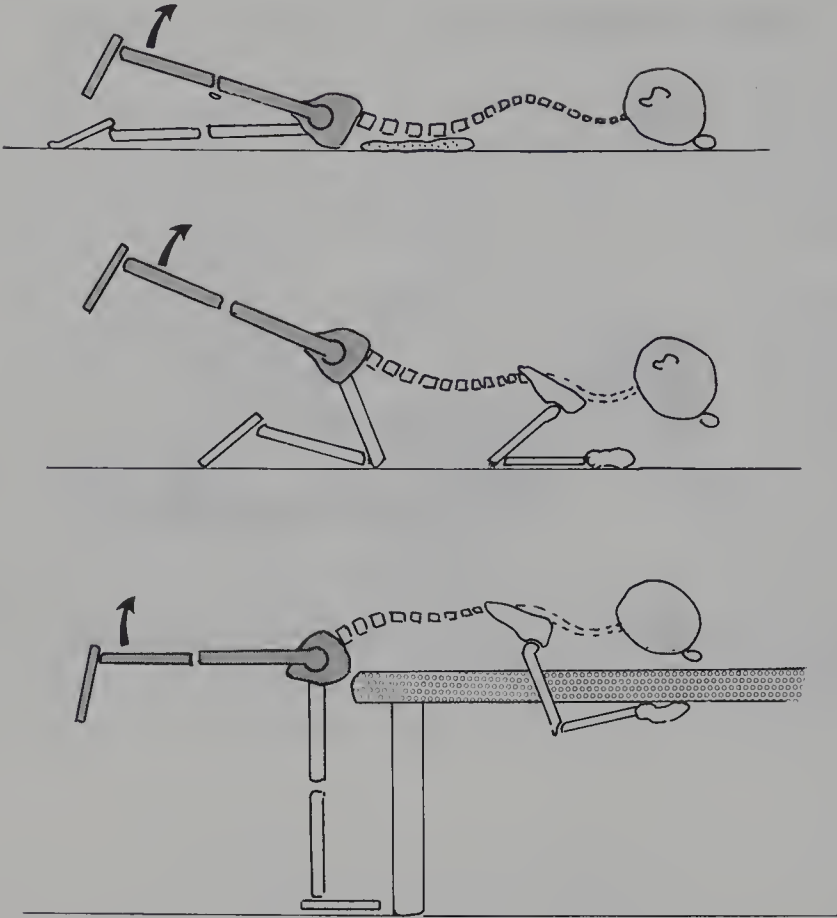


Figure 3-45. Hip extension test. This tests the range of motion of the hip flexors for flexibility. It can become an exercise to strengthen the extensor with added weights.

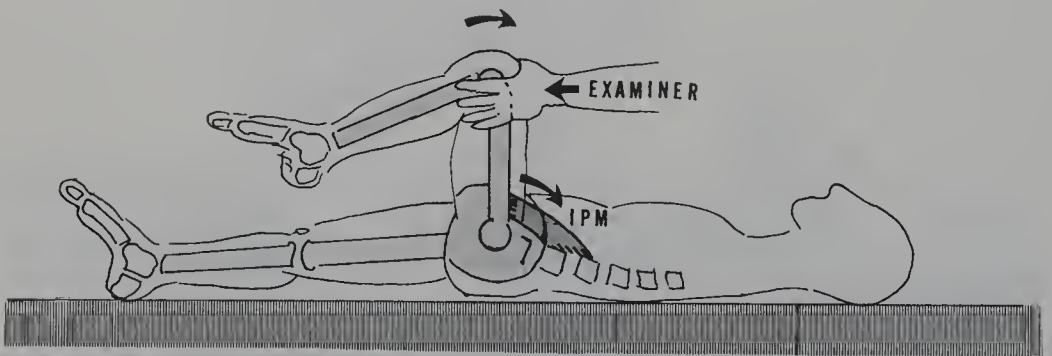


Figure 3-46. Hip flexor strength test. With the patient supine the hip is fully flexed (curved arrow) against the restraining hand of the examiner who can determine the strength and the endurance of the iliopsoas muscle (IPM).

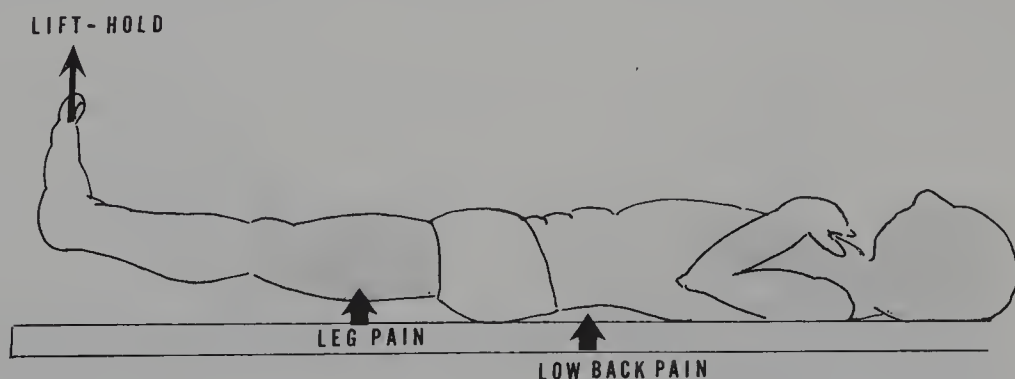
Exercises to correct or ameliorate the deficiency found from these tests are valid for general conditioning, but as a specific remedy of chronic low back pain, they are unproved.

## TESTS TO INCREASE INTRATHECAL PRESSURE

Admittedly objective and reproducible, these tests are also dependent upon verbal reports by the patient, which incur subjectivity. They are based on the fact that increased intrathecal pressure increases the pressure upon the involved and obstructed nerve root. They are more objective when used to confirm leg pain as emanating from the low back (see Chapter 4).

### Milgram Test

With the patient lying supine on the examining table, both legs are attempted to be held approximately 2 inches from the table for 30 seconds (Fig. 3-47).<sup>70</sup> If no pain occurs, there is no intrathecal pathology, whereas if pain does occur, pathology can be considered to occur. What pathology must then be determined. It must be remembered that this maneuver also causes a stressful hyperextension in the presence of weak abdominals and thus is a mechanical test not involving intrathecal pressure.



**Figure 3-47.** The Milgram test. This test, when considered positive, originally indicated increased intrathecal pressure. It requires the patient holding both legs some inches from the table for 30 seconds. This test also causes low back pain from lumbosacral hyperextension in patients with subnormal abdominal musculature but this is not a positive response.



## The Naffziger Test

Another test that increases the intrathecal pressure is the Naffziger test, in which the jugular veins are compressed gently for approximately 10 seconds until the patient's face flushes (Fig. 3–48). Then the request to cough should produce pain if there is increased pressure. In the presence of discogenic nerve compression, this test can be considered objective; however, it is also present in other cord and nerve compressive syndromes.

## The Valsalva Maneuver

The patient is asked to bear down as if to move the bowels or strain while holding one's breath. This maneuver increases intrathecal pressure (Fig. 3–49). It is positive if this maneuver produces pain in the low back or down the leg and thus becomes an objective test, but it does not signify only disk pathology but any other space-consuming thecal pathology.

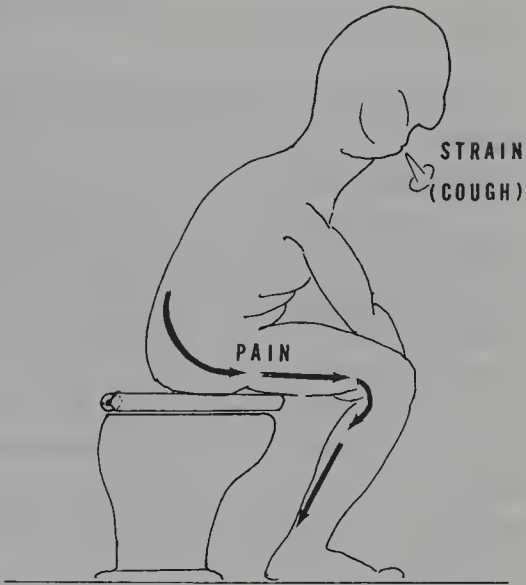
## The Kernig Test

The supine patient's head is forcefully flexed (Fig. 3–50) upon the chest, which allegedly stretches the spinal cord dura, including that distally covering the nerve roots. It is a modification of the well leg SLR test previously described. It may be positive from pathology or cord compression any way along the cord, thus it is not specific for lumbosacral pathology.



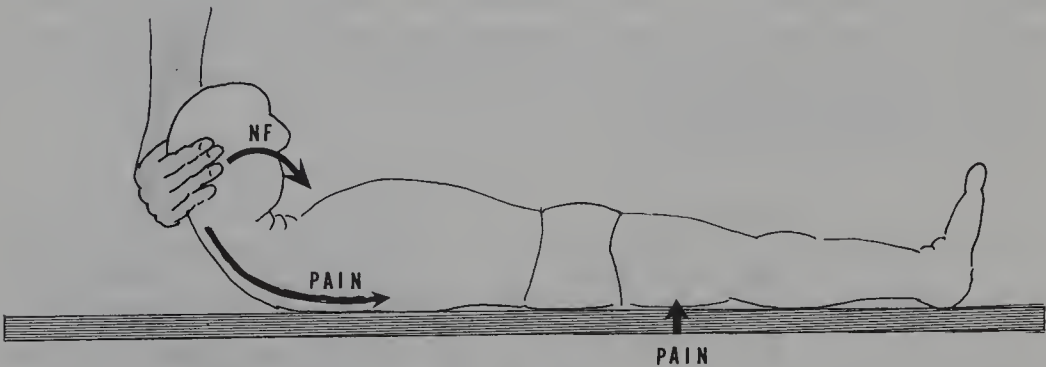
Figure 3–48. The Naffziger test. Compression of the jugular vein until the patient's face begins to flush causes an increase in intrathecal pressure if accompanied by a cough.

**Figure 3-49.** The Valsalva maneuver. When the patient bears down, as in moving the bowels or coughing, there is an increase in intrathecal pressure. If any of these maneuvers cause posterior leg pain it indicates pressure on the theca somewhere in the cord.



### Confirmatory Tests for Sciatic Radiculitis

An electromyography examination will give objective evidence of nerve root compression, demyelination, or inflammation. X-rays, CAT scans, and MRIs also confirm compression on nerve roots. These tests will be further discussed in Chapter 5.



**Figure 3-50.** The Kernig test. With the patient supine the head is forcefully flexed to bring the chin on the chest. If pain radiates down the spine and into the leg it is an indication of dural irritation or compression somewhere along the cord or pathologic abnormality within the cord. The clinical examination confirms the site of cord (dural) compression.

## Tests for Malingering

There are tests designated to determine whether the patient is malingering, even though there is disagreement as to malingering being a psychological aberration.

The problem relates to whether the patient is providing inaccurate information consciously and intentionally. The definition of malingering is "deliberate faking of symptoms for the sole purpose of obtaining an extrinsic goal."<sup>70</sup> The frequency of this accusation varies from frequent<sup>71</sup> to rarely.<sup>72</sup>

There is no good marker that correlates the objectivity of findings with the subjectivity of the incurred disability.<sup>73</sup> The major consensus among examiners is exaggeration of symptoms and inconsistency such as weakness to manual testing not consistent with the effort expected (Fig. 3-51).

Numerous tests have been devised to determine the presence of malingering such as neural blockade, and so on, but all have deficiencies.<sup>74</sup> The "diagnosis of malingering should not be reached lightly nor be based on a few inconsistencies between verbal reports and actual behaviors."<sup>75</sup>

In summary the number of objective clinical tests to determine nonorganic signs to validate impairment are few if even existent, thus their performance in performing an examination should be interpreted with care and with question as to their being the one cause or one of several causes in the patient's complaint. This inadequacy also presents a question as to the manner in which treatment protocols are evaluated as being effective.<sup>76</sup>

With the current excessive expenditure incurred in evaluating low back pain, especially chronic low back pain, a careful review of the physical examinations being performed to validate subjective complaints must be done. Their performance may be standardized, but their interpretation remains subjective and dependent upon examiners' experience, which is also subjective.

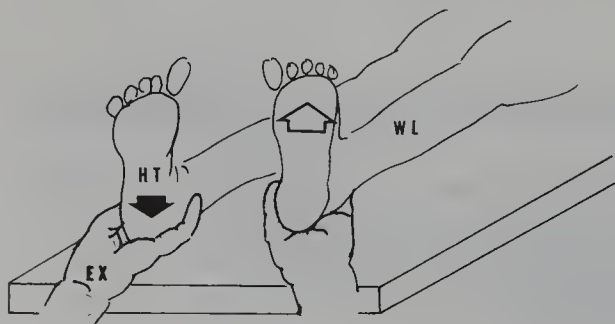


Figure 3-51. The Hoover test. The supine patient who claims a weak leg (WL) when asked to hold up that leg should push down with the opposite leg (HT) against the examiner's hand (EX). Failure to push down with the opposite leg is considered a positive Hoover test.

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## CHAPTER 4

# Musculoskeletal Pain

Pain in the low back is a leading reason for patient visits to physicians, for hospitalizations, and for work disability. Statistics indicate that 60% of the population in industrialized nations suffer from low back pain at some time while working<sup>1</sup> and each year 5% of American adults experience an episode of low back pain.<sup>2</sup> Of the 7 million new cases of low back pain, 5 million will become partially disabled and 2 million remain unable to function.<sup>3</sup>

In spite of these facts, there appears to be a paucity of valid evidence in the literature and in clinical practice that supports most current methods of objectively evaluating or realistically treating spine disorder.<sup>4</sup>

Most clinicians interpret the symptoms and impairment of low back disorders as a biomechanical disorder.<sup>5</sup> With acceptance of this concept, there has resulted a high success rate in treating acute low back pain,<sup>6</sup> but a low rate of success in treating chronic low back pain.

The tissue sites of pain in the low back have been documented, and in subsequent chapters each will be individually discussed.<sup>7</sup> These include discogenic pain, facet pain, and, in clinical practice, muscle pain, muscles being the most frequent site of nociception.<sup>8</sup> Muscle pain has been thoroughly discussed in Chapter 1, to which the reader is referred. The method of specific examination has been discussed in Chapter 3.

The roles that muscles play in the control of trunk motion and in posture have been discussed. As Ladin says, "Muscles balance the external movements" in response to external loads.<sup>9</sup> The other vertebral and paravertebral structures equilibrate the external forces and balance the muscle forces. "The smallest possible muscle forces that satisfy the moment equilibrium equations and minimize the muscular spinal com-

pression force” are the physiological needs of activity.<sup>9</sup> Violation of this adage, is the basis of low back pain and dysfunction.

## FATIGUE

Human motor performance is influenced by fatigue. Fatigue has been variously defined<sup>10</sup> but has been aptly called<sup>11,12</sup> “a transient loss of performance capacity resulting from preceding performance regardless of whether the current performance is affected.”

Fatigue can be based on two anatomic sites: (1) central fatigue within the central nervous system<sup>13</sup> and (2) peripheral fatigue, residing outside the central nervous system.<sup>14</sup> Peripheral fatigue probably is more in the realm of neuromuscular unit and has a chemical basis, whereas central fatigue is probably more in the motivational and psychological areas, as well as having neurophysiological aspects.

Neuromuscular transmission failure does not appear to be a major cause of fatigue for voluntary efforts and may be due largely to the slowing of motoneuron firing.<sup>14</sup> The chemical aspects of fatigue<sup>15</sup> have been postulated to be the rate of consumption of oxygen and the acidosis effect upon the brain contributing to the “sensation” of fatigue.<sup>15</sup> Studies in athletes have shown muscle fatigue to increase proportionately to the rate of depletion of muscle glycogen.<sup>16</sup>

Fatigue can be considered as contributing to ultimate low back pain as related to its effect on the proper neuromuscular mechanism and be involved in acute low back pain but cannot per se be considered to be a significant factor in chronic low back pain.

## FIBROMYALGIA

Many back pain patients frequently present with a generalized musculoskeletal syndrome, of which the low back site is prevalent. Patients having musculoskeletal symptoms utilize physician’s services at twice the rate of average primary care patients.<sup>17</sup>

The painful musculoskeletal system has increased the use of the term *fibromyalgia*<sup>18</sup> at an exponential rate in the medical literature.<sup>19</sup> There is a tendency now to differentiate fibromyalgia as *primary* or *reactive*, with the latter having a preceding trauma resulting in the painful muscle group(s). This is a deviation, as trauma was to be excluded to confirm the diagnosis of fibromyalgia.<sup>20</sup>

As I have written earlier, the condition of fibromyalgia is “a syndrome characterized by chronic pain widely distributed through all the

skeletal muscles and soft tissues.”<sup>21</sup> The International Association for the Study of Pain has generalized the diagnosis under the heading of primary fibromyalgia syndrome (PFS)<sup>22</sup> and classified these chronic musculoskeletal pain syndromes “without identifiable cause” as:

1. *Primary fibromyalgia syndrome (PFS)*: fibrositis or diffuse myofascial pain syndrome; primary diffuse fibrositis syndrome
2. *Myofascial pain syndrome (MPS)*: specific myofascial pain syndrome
3. *Temporomandibular pain and dysfunction syndrome (TMPDS)*

The diagnostic criteria of fibromyositis are based on subjective long-standing symptoms of musculoskeletal pain, fatigue, sleep disturbance, and clinical findings of reproducible tender points.

The general diagnosis of PFS is that of Yanus,<sup>20</sup> later modified by Smythe,<sup>23</sup> which probably does not directly relate to low back pain syndromes but must be addressed, as low back pain has been considered a reactive fibromyositis in recent literature.

The “obligatory” criteria to diagnose PFS are:

1. Generalized aches and pains or prominent stiffness, involving three or more anatomical sites for at least 3 months’ duration
2. Absence of traumatic injury, structural rheumatic disease, infectious arthropathy, endocrine-related arthropathy, and abnormal laboratory tests

With these two criteria needed for diagnosing PFS, it is difficult for the author to entertain the diagnosis of fibromyositis as the etiology of low back pain resulting from a traumatic episode, yet that is what the proponents of reactive fibromyositis would categorize as involved in chronic low back pain following an industrial or vehicular accident.

The criteria are further delineated as:

Major:

1. Three or more typical and consistent tender points

Minor:

1. Modulation of symptoms by physical activity (or)
2. Symptoms altered by weather changes
3. Symptoms aggravated by anxiety or stress



4. Poor sleep
5. Generalized fatigue or tiredness
6. Anxiety
7. Chronic headaches
8. Irritable bowel syndrome
9. Subjective swelling
10. Numbness: nonradicular and nondermatomal

Smythe<sup>23</sup> modified the criteria as follows:

1. Widespread aching of more than 3 months' duration
2. Local tenderness at 12 to 14 specific sites
3. Skin rolling tenderness over the upper scapular region
4. Disturbed sleep, with morning fatigue and stiffness
5. Normal erythrocyte sedimentation rate (ESR), serum glutamic-oxaloacetic transaminase (SGOT), rheumatoid factors test, anti-nuclear factor (ANF), muscle enzymes, and sacroiliac films

Sleep impairment has been confirmed by sleep laboratory findings in patients diagnosed as having FBS and patients developing symptoms by sleep deprivation.<sup>24</sup> A century ago the American psychiatrist George Miller Beard described a syndrome of generalized fatigue, widespread pain, and psychological disturbance as "neurasthenia" and attributed it to "the stress of modern life,"<sup>25</sup> and in 1913 the syndrome of *fibrositis* was coined.<sup>26</sup> In 1942 a Lancet editorial stated, "The condition referred to as fibrositis is still one of vague pathological and clinical definition: some sceptics deny its existence altogether, so that is it not surprising that the diagnosis has fallen into disrepute."<sup>27</sup>

Kelly in 1946 concluded that "fibrositis has not a single cause but results from combined action of many factors, some known and others unknown."<sup>28</sup> There has been little verification of this diagnosis since, yet it is being offered to explain persistent impairment and disability at a tremendous expense to the society of today and a significant financial benefit to today's medical and legal organizations.

The questions remain: Is fibromyalgia a muscle disease? Is fibromyalgia a psychiatric disease? Is fibromyalgia a neuroendocrine illness? Is fibromyalgia the master of disguise?<sup>29</sup> There are too many patients throughout the world suffering from this syndrome to completely dismiss this diagnosis, so the plea can be offered that "further studies, based on scientific foundation, will give greater reality to that which remains obscure."<sup>29</sup>



## PATHOPHYSIOLOGY

Abnormalities of the musculature in this syndrome have been implicated, with some reporting abnormal histological changes, but none remains confirmed or reproducible. The muscle pain of fibromyalgia is similar to the muscle pain of benign low back pain, and in that context both can be discussed.

### Pathophysiology of Fibromyalgia

Yanus speculated that all the histological abnormalities noted could result from microspasm, causing ischemia of the musculature.<sup>30</sup> The algogens produced by trauma all affect the local microcirculation (see Fig. 1-73), causing vasoconstriction or vasodilation and also increasing vascular permeability with resultant extravasation and edema (see Fig. 2-11).

Ischemia of the involved tissues results in pain, which reflexively causes spasms of the contiguous tissues (Figs. 4-1 and 4-2).

The hypoxia of the muscle allegedly decreases the intramuscular level of adenosinetriphosphatase and phosphocreatine, which accounts for the pain.<sup>31</sup> Compromised capillary microcirculation in a fibrositic trapezius muscle has been found to be diminished after the application of ultrasound as compared to a normal muscle.<sup>32</sup>

Electromyographic studies have failed to reveal specific abnormalities in fibromyalgia, although fatigue in fibrositic muscles has been demonstrated.<sup>33</sup> Impaired muscle strength has been claimed in fibrositic patients as evaluated by Cybex II dynamometer,<sup>34</sup> but the objectivity of this conclusion has been questioned as to whether the maximum effort was made in the presence of pain.<sup>35</sup>

The neurophysiology of the sleep disturbance noted in fibromyalgia has not been clarified.<sup>36</sup> Depletion of serotonin has been postulated in non-REM sleep.<sup>37-39</sup> Serotonin is present in platelets and is released after trauma. It is a potent vasoconstrictor and thus is related to the ischemic pain of fibrositis syndrome patients. It also affects sleep. The relationship of sleep deprivation in fibromyalgia and its relief from tricyclic medication has enforced the relationship of serotonin as tricyclic medications block the re-uptake of serotonin at the synaptic cleft.<sup>39</sup>

The factor of diminished levels of tryptophan in fibromyalgia has been expounded,<sup>40</sup> but its value in the therapy has been questioned.<sup>41</sup>

Fibrositis syndrome patients have been found to have higher urinary norepinephrine levels than normal.<sup>42</sup> This finding is consistent with patients having high levels of anxiety but was not found in patients

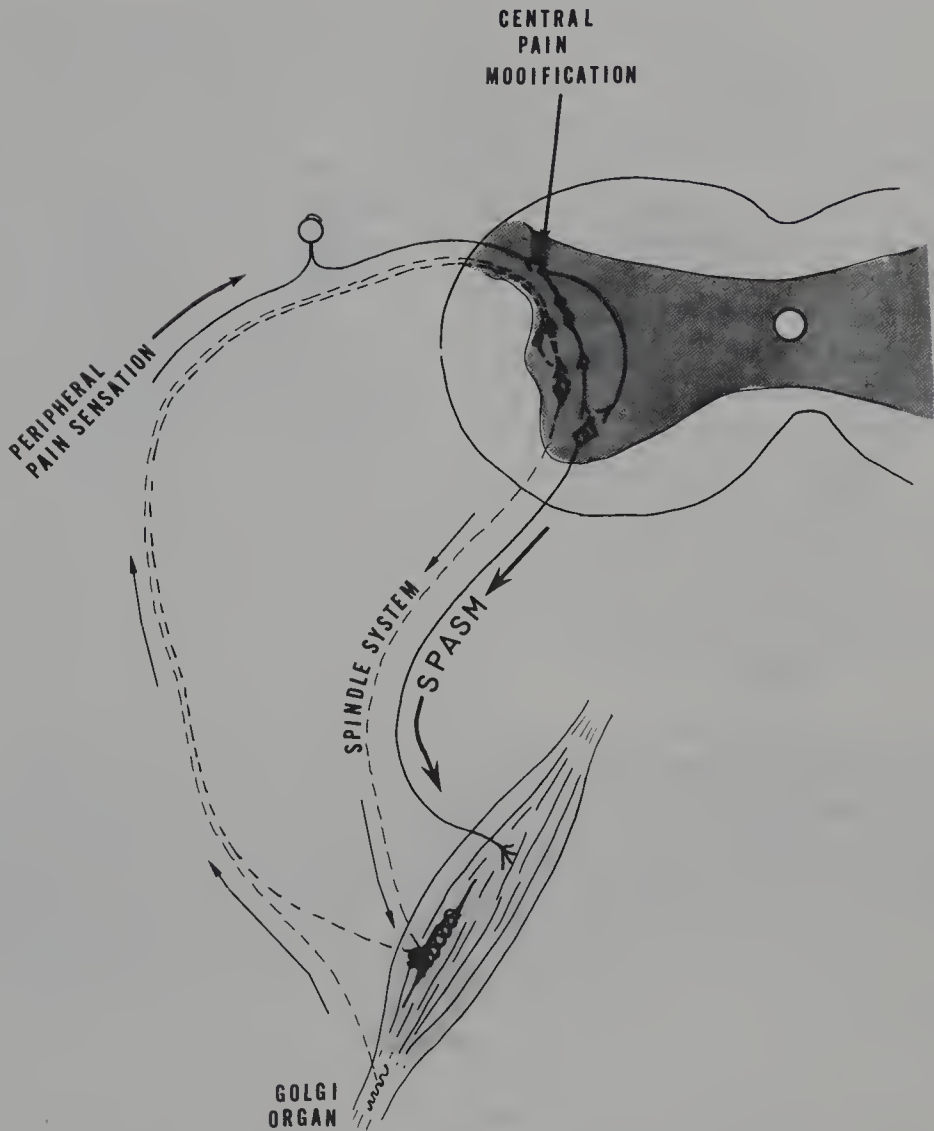


Figure 4-1. Neural patterns producing pain with afferent impulses ascending to flexed layers of the dorsal horn have a mononeuronal connection to the ipsilateral anterior horn cells that innervate the segmental muscles, causing a contraction of the extrafusal fibers. The spindle system is also innervated and reset.

exhibiting depression. In cases clinically diagnosed as fibrositis syndrome, patients were found to have high levels of substance P in their cerebrospinal fluids, but this was not compared to other painful neuromuscular conditions.<sup>43</sup>

Physical fitness, which allegedly decreases the perception of pain,<sup>44</sup> was found to be ineffectual in fibrositis syndrome patients.<sup>45</sup>

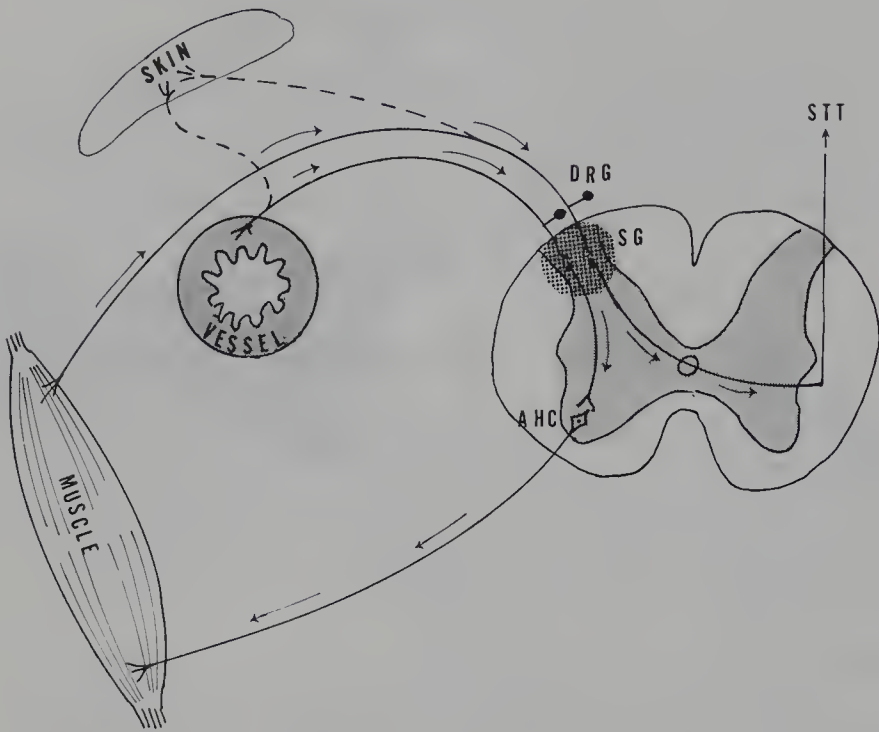


Figure 4-2. Neural patterns emanating from the skin, blood vessels, and muscles causing spasm. The nociceptive substance liberated from a muscle in a sustained contraction (spasm) ascends through the dorsal root ganglion (DRG) with further transmission into the substantia gelatinosum (SG) (Rexed layers II) then traverses to the spinothalamic tract (STTs) and ascends to the thalamus. A small branch traverses to the anterior horn cell (AHC), which causes further muscle contraction. The involved blood vessels and overlaying skin enhance the pattern.

The pathophysiology obviously remains unconfirmed,<sup>46</sup> but current concepts propose the syndrome to be a “complex interaction of nociceptive and neuropathic with dysregulatory central nervous system function enhanced by psychosomatic mechanisms.”<sup>47</sup> This concept, albeit intriguing, places the syndrome in the psychoneuromuscular area, which is equally vague.

## Pathophysiology of Muscle Pain

Muscle pain from the numerous etiologies of low back pain other than fibromyalgia remains to be addressed. The neurophysiological basis of muscle pain is essentially increased impulse activity of its nociceptors. The basis for this increased activity is probably an increase in

the chemical substances generated by muscles that have been overused, misused, abused, or traumatized.

The afferent fibers existing in muscle that are considered as transmitters of nociceptive impulses are thin myelinated fibers (A delta or group III) or unmyelinated fibers (C or group I-IV). The nerve endings were considered to be free endings lacking corpuscular receptive structures,<sup>48</sup> but this has been refuted in the finding that all free nerve endings are ensheathed by Schwann cells, leaving only small portions uncovered, where they are exposed to interstitial fluids.<sup>49</sup> These uncovered sites are considered to be the sites where external stimuli act. The typical location of these endings are the walls of the arterioles and the surrounding connective tissues. The precise nerve type, nerve ending, and ultrastructure in nociceptive transmission remain unclear.

**Neuropeptides of Afferent Fibers.** Neuropeptides are considered to be the transmitters of sensory information within nerve structures. The neuropeptides from muscle afferent fibers within dorsal root ganglia are similar to cutaneous and visceral nerves and have not been differentiated with afferents from muscles, but recent studies implicate a calcitonin-gene-related peptide (CGRP).

Substance P (SP) and its relationship to CGRP has been evoked as pertinent in muscle pain. Its action is vascular, which has been considered prominent in "neurogenic inflammation."<sup>50</sup> Muscle that is contained within fascia results in increased interstitial pressure, causing mechanical cell damage. The resultant ischemia is also nociceptive. Nociception has also been attributed to thermal and mechanical as well as chemical stimuli.<sup>51,52</sup>

Other chemical stimulants that have been considered nociceptive are bradykinin (BKN), 5-hydroxytryptamine (5HT), and high concentrations of potassium.<sup>53-55</sup> BKN is cleaved from a plasma protein precursor, and 5HT is released from platelets after vascular damage.

The above analgesic substances released from trauma have been shown to have a potentiating effect at the nociceptor sites.<sup>8,56</sup> When they are preceded by serotonin or prostaglandin E<sub>2</sub>, the effect upon the receptor of bradykinin is enhanced by 10 minutes. These substances affect local microcirculation, causing vasoconstriction or vasodilation but also increasing vascular permeability, which results in extravasation and edema. As has been stated, in fibrositis syndrome another effect of the liberated algogens is reflex local muscle spasm, which further enhances local ischemia.

An unexpected finding was the difference of receptors within different portions of muscle.<sup>57</sup> Further confusing the picture of muscle pain is the finding of silent receptors, which cannot be activated by mechanical means under normal circumstances but respond readily when preceded by inflammation.



Specificity of sensory afferent nerves has long been postulated. This specificity postulates that specific nerves transmit specific sensations and thus that activation of a specific nerve ending always elicits the same sensation regardless of the stimulus. This concept also postulates that receptors are polymodal: serving ergoreceptive as well as nociceptive functions. The sensitivity of muscle receptors is essentially modulated by numerous chemicals, but all neurotransmission is similar.

The treatment of muscle pain thus is chemical-mechanical intervention that modulates the sensitivity of nerve transmission.

## CAUSATION OF MUSCLE PAIN

Trauma is undoubtedly the major cause exciting activation of muscle nociceptors and results in muscle pain from: (1) broken fibers leaking potassium into the interstitial fluid; (2) blood extravasation from damaged blood vessels liberating BKN from plasma proteins, which in turn release prostaglandins; (3) neuropeptides (CGRP and SP) liberated at the nerve endings; and (4) vasodilatation causing mechanical compartment pressure.

Trauma can be considered as unaccustomed intensity or duration of muscle contraction, especially eccentric contractions.<sup>57</sup> Eccentric contractions (negative work), in which the muscle performs lengthening contractions (deceleration) produce external forces that exceed those produced within the muscle itself. Eccentric contractions allegedly activate a smaller number of motor units that are activated during positive contractions of the same intensity. The mechanical stresses upon the Z bands in the sarcomeres and the connective tissue are higher<sup>58</sup> and greater muscle damage has been documented.<sup>59</sup>

Lactate concentration, originally postulated from excessive muscular contraction, is now doubted, as there usually occurs a delay in pain after activity and lactate concentration has returned to normal after 1 hours of activity. Prostaglandin liberation after excessive muscular activity as a cause of muscle pain is also refuted, as administration of a nonsteroidal anti-inflammatory drug (NSAID) after eccentric activity is not effective.<sup>60</sup>

Ischemia, long considered a factor in muscle pain, remains controversial.<sup>61</sup> Interruption of blood flow in a resting extremity does not cause pain unless the muscle contracts during the ischemic condition. The causative substance appears to be BKN, which is released from plasma proteins,<sup>62</sup> which strongly sensitizes nociceptors in the muscle. Use of a proteinase inhibitor, which prevents the release of BKN, allows patients with intermittent claudication to walk without pain.<sup>63</sup>

Tension or sustained muscle tone has been implicated in muscular



pain, although muscle tone is not well understood. A completely relaxed muscle is electromyographically silent, assuming therefore that tonus is viscoelastic (osmotic fluid pressure, elastic tone of connective tissue, including fascia) in nature.

Sustained muscle tone during mental or emotional stress may be painless and can be abolished by voluntary relaxation. Cramps and spasm are sustained, involuntary muscle contractions that cannot be released voluntarily and electromyogram (EMG) activities are evident.<sup>64</sup>

Spasm pain cannot be attributed to increased muscle tension, as the intramuscular pressure is insufficiently elevated. Pain may occur from contraction under an ischemic situation as recent studies have indicated an increase in the  $PO_2$  inside the muscle in spasm.<sup>65</sup>

A neurological reflex mechanism has been postulated in which the alpha motoneurons are activated in the cord (Figs. 4-3 and 4-4). This cycle, however, is functional only in flexor muscles, while extensor muscles<sup>66</sup> are inhibited only by activation of nociceptors of the homologous\* muscle. Activation of the extensor muscles by activation of the gamma loop remains to be explained.

The gamma loop has been well documented in neurophysiology.<sup>35</sup> It is a cycle between the interfusar system and the extrafusar fibers as well as the relationship between agonist and antagonist innervation. The gamma system is also activated by the descending fibers of the extrapyramidal motor pathways<sup>35</sup> and are thus influenced by psychic stresses. This mechanism may well explain some of the relationships of painful muscle tension in patients with psychic stress.

In a discussion of low back pain as related to erector spinae muscle tension, there are obviously many aspects to be considered. The causation of low back pain, including discogenic disease, has implied that faulty neuromuscular activity can cause functional units irritation of nociceptor tissues. Once nociception has been initiated, which tissues persist in emanating pain remains conjectural, but muscle, especially the erector spinae, has been implicated.

To diagnose the condition as reactive fibromyositis is simplistic yet the clinical condition merits consideration. Diagnosis also imputes taxonomy of the basic condition, further confusing diagnostic criteria. Treatment based on appropriate diagnosis is also jeopardized as the exact causation and manifestation of the involved tissues must be addressed.

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\**Homologous*: Similiar in fundamental structure and in origin but not necessarily in function. (From Thomas, CL (ed): *Taber's Cyclopedic Medical Dictionary*, ed 16. FA Davis, Philadelphia, 1989, with permission.)

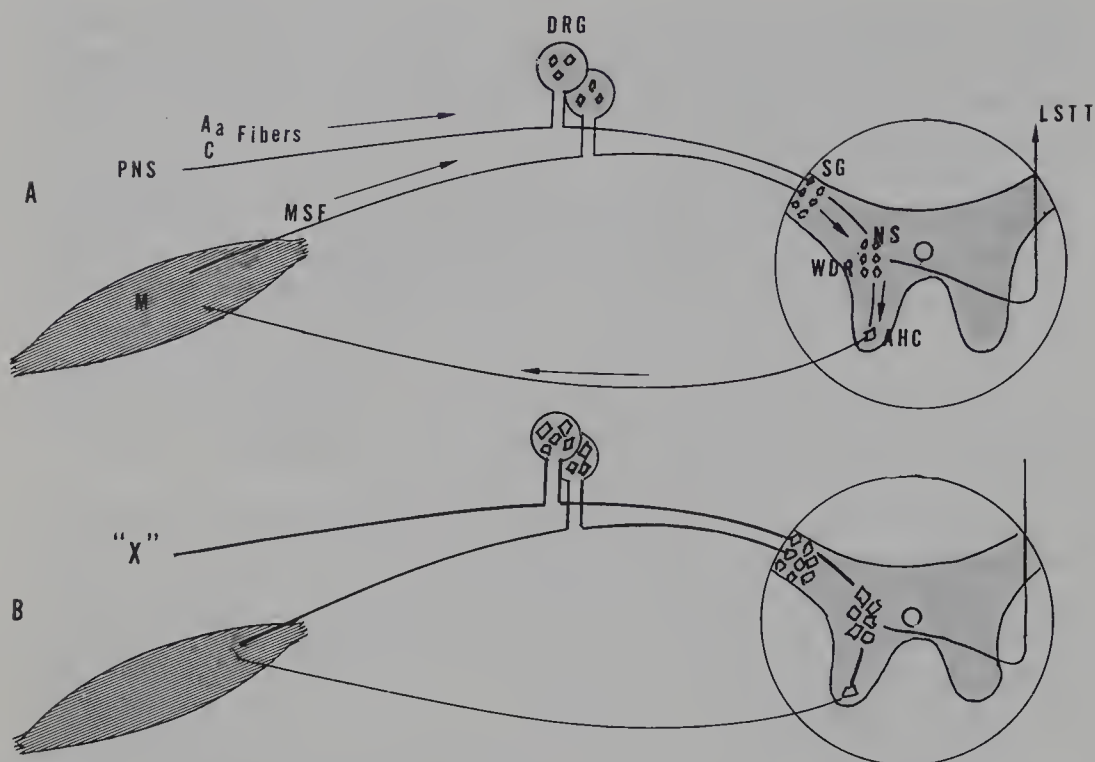


Figure 4-3. Expanded fields of hyperalgesia. The accepted route of nociception from peripheral sites (PNS) via the A alpha and C fibers through the dorsal root ganglion (DRG) into the dorsal horn of the cord, into Rexed layers I and II (substantia gelatinosa [SG]) (A). The nociceptive impulses stimulate nociceptive specific neurons (NS) with stimulation of wide dynamic range neurons (WDR). Ultimately these nociceptive impulses ascend to the limbic system then to the cortex via the lateral spinothalamic tracts (LSTT). It is perceived at the cortex as pain. Muscle sensory fibers (MSF) also ascend to the DRG then along similar pathways as the ascending sensory fibers. Hyperalgesia results from intense, continuous or repeated nociception at the periphery (X) (B). These barrages increase the number and sensitivity of the nerve cells within the DRG and the SG, increasing the sensitivity of the entire pathways. This postulated mechanism explains why impulses not specifically nociceptive can increase the irritability of the painful area.

Methods of documenting functional and severity status in fibromyalgia have not been validated.<sup>36</sup> Chronic pain emanating from the low back also intervenes in restoring pain-free function and must be addressed. Strengthening exercises in treating chronic low back pain have thus far been disappointing, and subjects treated with lumbar extension exercises have admittedly improved in strength and have experienced some reduction in pain, but these exercises did not materially result in improved activities of daily living.<sup>37</sup> Disability established from the impairment remains a major concern.

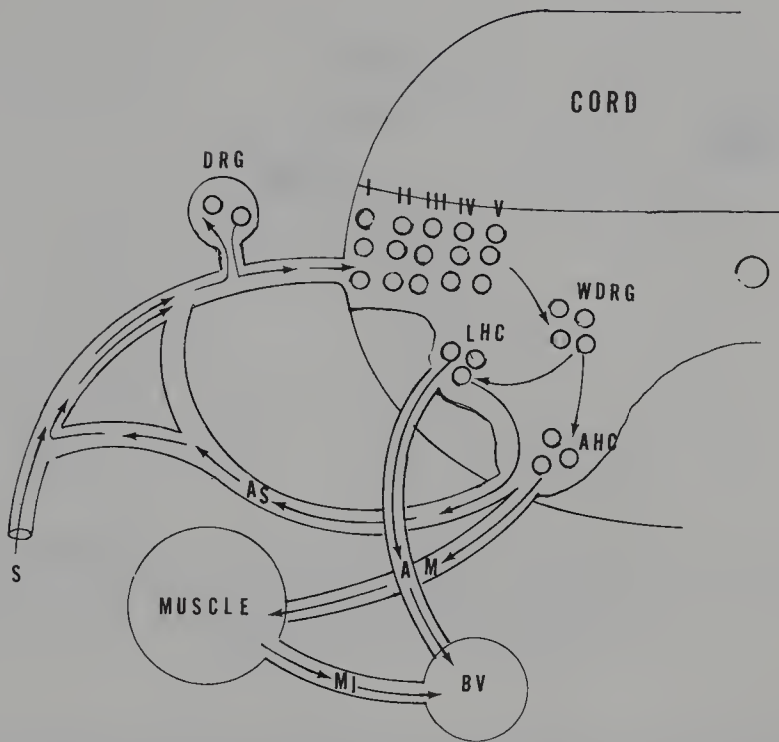


Figure 4-4. Neurologic pathways of nociception. The sensory impulses (S) enter the dorsal root ganglion (DRG) via afferent fibers of unmyelinated and sparsely myelinated fibers (C and A alpha). They proceed to the Rexed layers I and II, which are the substantia gelatinosa. Interneuronal fibers transfer impulses to the lateral spinothalamic tracts (not shown), which ascend to the hypothalamus, the thalamus, and to the cortex. Fibers transmit impulses from the Rexed layers to the dynamic wide range fibers (WDRG) with interneuronal fibers transmitting to the anterior horn cells (AHC) and fibers to the lateral horn cells (LHC). The latter activates muscle activity (M), the former activates autonomic impulses that innervate blood vessels (A). Afferent impulses are transmitted (AS) to the DRG. Impulses from the muscles that are excessively contracted (spasm) are transmitted to the blood vessels (MI), causing vasoconstriction.

The theories regarding muscular contraction in the mechanisms of low back pain are still unclear, but it can be concluded that muscles are involved in a major way. Spinal function is related directly to neuromuscular function<sup>67</sup> and neuromuscular malfunction results in pain from irritation of the tissues that are the nociceptors causing pain (Fig. 4-5).

The muscle component is a primary consideration in the acute low back pain, but it is also a factor in chronic low back pain. The psychological aspect of low back pain (Chapter 2) also involves muscle in the mediation of pain.

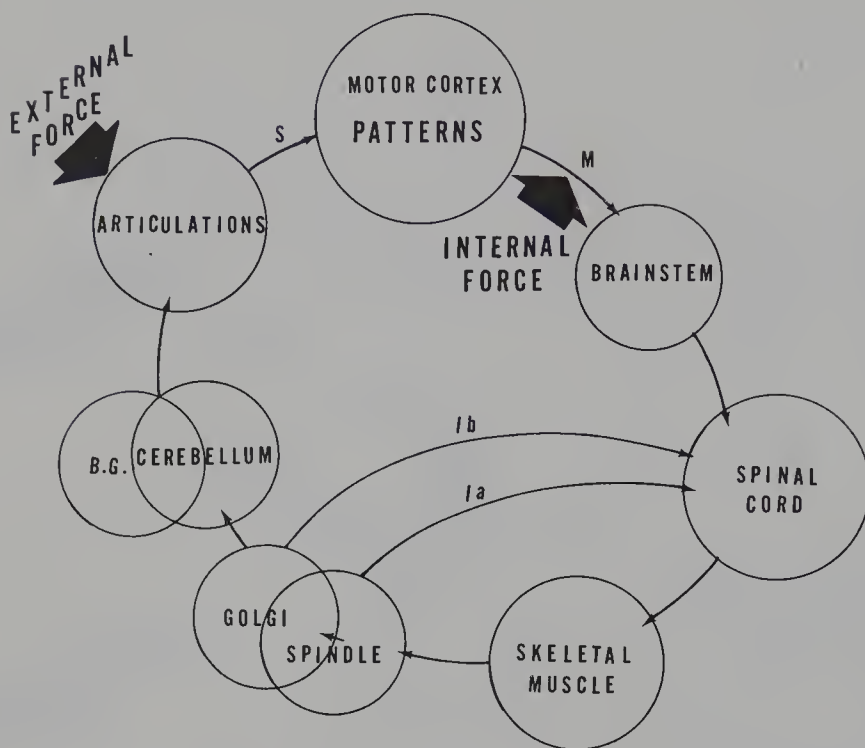


Figure 4-5. Motor cortex patterns. The motor patterns originate in the cortex and are transmitted through the brainstem to the spinal cord from where the muscles that activate the body are stimulated. The ultimate neuromuscular activities are coordinated by the Golgi and spindle systems within the muscles. The central neuromuscular system is coordinated by the basal ganglia (BG) and the cerebellum. The articulations also send sensory (S) impulses to the cortex to inform that the intended action has been performed.

Unlike cutaneous pain, usually characterized as sharp, stabbing or burning, muscle pain has a dull, aching characteristic.<sup>68</sup> In contrast to sensory pain, which can be localized with great accuracy, muscle pain is diffuse and ill defined.

Muscle pain, like visceral pain, is often associated with autonomic symptoms such as drop in blood pressure, sweating, and nausea.<sup>69</sup>

The dorsal horns that process the neural information from muscles receive additional input from cutaneous receptors.<sup>70,71</sup> Recent data indicate that information from muscle and cutaneous nociceptors is processed differently in the spinal cord.<sup>72</sup> This difference is provocative as muscle pain is subject to a stronger descending inhibition, as are cutaneous nociceptors,<sup>73</sup> and implies the basis for muscle pain being so difficult to evaluate and manage.

Muscle pain implies origin of nociception from striated muscle but also from its fascia and tendinous insertions. Muscle nerve fibers in the



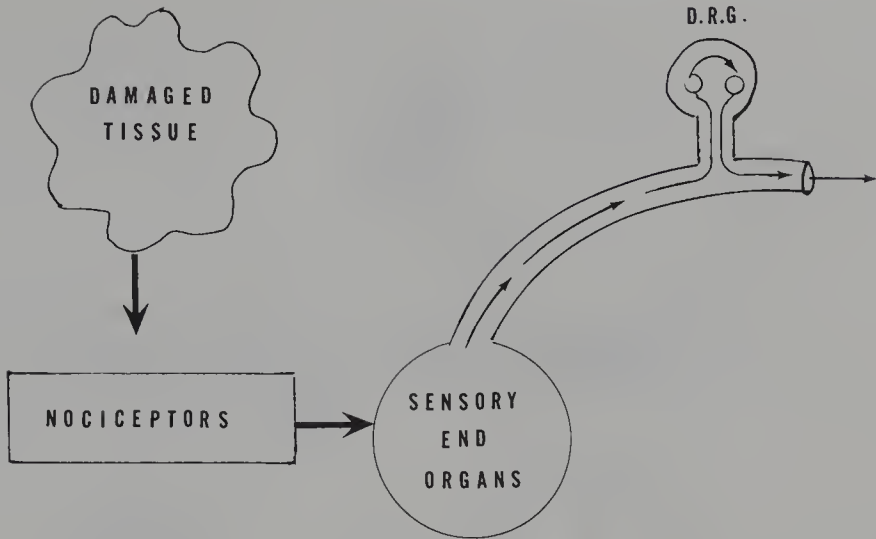


Figure 4-6. Damaged tissue activating nociceptors. The damaged tissue liberates nociceptive substances that irritate the nerve endings (nociceptors), which transmit impulses to the dorsal root ganglion (DRG) nerves.

cat were composed of one third myelinated and two thirds unmyelinated with 50% of the latter being sensory, and of these, 43% nociceptive.<sup>74</sup> Admittedly these studies were in cats and even in that species various muscles varied; therefore, these findings cannot be interpolated in humans.

Muscle afferent fibers, including nociceptive ones, terminate in laminae I, II, and IV in the dorsal horn, and as they have been activated by stimulation of muscle fibers, it can be assumed that specific nociceptors are present in skeletal muscle and that specific fibers end in specific areas of the dorsal horn.

The precise nociceptive mechanism at the end organs remains unconfirmed (Fig. 4-6). Accumulation of lactate, potassium ions, and/or metabolic products from ischemia have been proposed but not confirmed. Weak mechanical stimuli and muscle contraction have been recorded and are currently termed ergoreceptors, but their role in pain remains obscure. Dorsal horn neurons responding exclusively to activation of muscle nociceptors are extremely rare in the cat or the rat as most are convergent impulses with cutaneous stimuli and their presence in the human also remains unconfirmed.

The pain-spasm-pain feedback circuit (Fig. 4-7) remains conjectural and speculative. Immediate pain from acute muscle trauma is probably caused by *direct mechanical activation* and the ensuing tenderness by sensitization of the receptors by endogenous substances released by the trauma. Muscle soreness is probably caused by histological alterations of the fibers, such as fiber tearing and/or cellular infiltration.



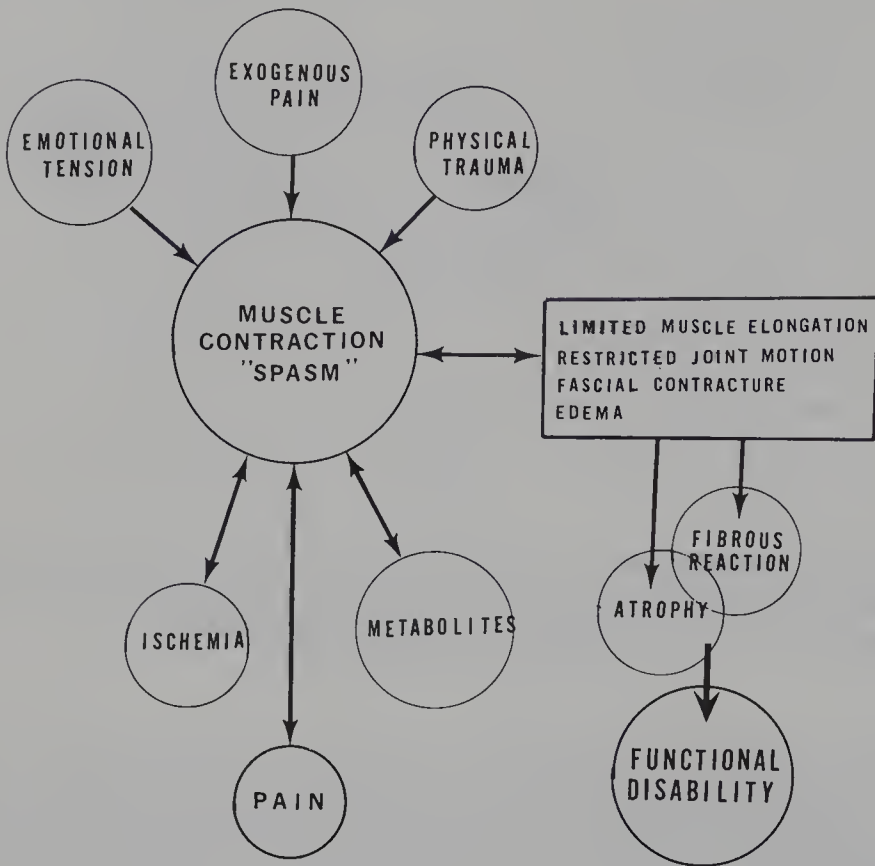


Figure 4-7. Muscle spasm and functional disability. The sequence is self explanatory. Trauma, emotional tension, exogenous pain, and physical trauma, causing ischemia from vasoconstriction and edema, elicit muscular spasm. Prolonged or excessive spasm causes limited active motion with subsequent edema and fascial contracture. Muscular atrophy and articular fibrous reaction lead to impairment and functional disability.

Activation or sensitization of the receptors may be enhanced via the ischemia from contracted muscle.

The ascending pathways may be further enhanced via the extrapyramidal motor pathways by chronic psychic stress.

It is apparent that current knowledge of the pathophysiological mechanisms of muscle pain, which is so pervasive clinically, is woefully incomplete yet is sufficient to encourage meaningful therapeutic intervention at all levels and stages (Fig. 4-8).

Low back pain from abnormal, excessive, or inappropriate activities, either external or internal, can cause excessive neuromuscular reaction, which causes pain and ultimately may cause impairment or disability. The external forces may be direct body trauma, such as a fall,

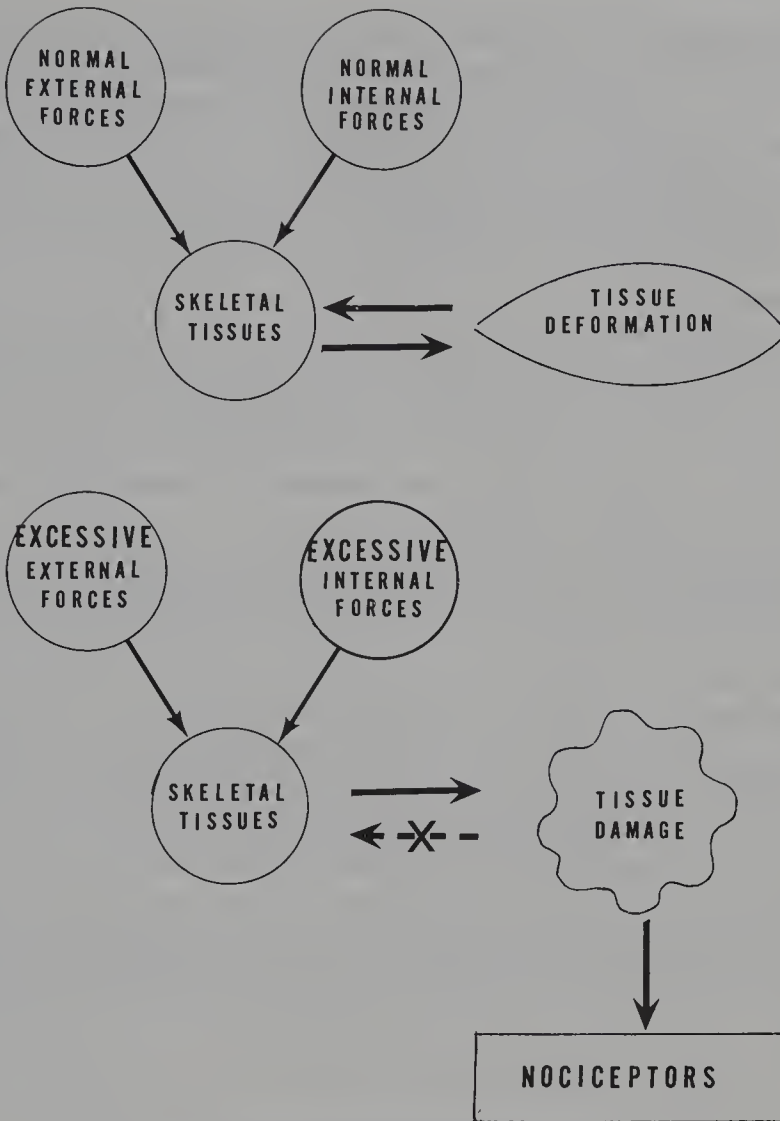


Figure 4-8. Tissue deformation versus tissue damage. External and normal internal forces imposed on skeletal tissues cause a reversible deformation as long as the tissues are not structurally damaged. When the forces, external or internal, are excessive they exceed the physiologic limits of the tissue with resultant tissue damage that is often irreversible.

an athletic impact, or a vehicular accident. Internal forces are inappropriate activities that misuse the muscles, with resultant injury to the joints (facets), ligaments, muscles, and/or the disk tissues.

If the forces are acceptable and essentially within physiological limits, the tissues may be temporarily deformed yet recover without permanent structural deformity or damage. Pain results with limited disability but no impairment.

Failure to properly address these tissue injuries may lead to iatrogenic complications, permitting disuse, atrophy, and psychological impairment. The word *iatrogenic* implies that unnecessary disability and impairment is the fault of the physician, caused by the failure to perform an adequate examination, the ordering of inappropriate therapy, or the failure to involve the patient in understanding the extent and features of the impairment, allowing further disuse and psychological deterioration.

The muscular system is a major concern in this aspect of lumbosacral pain. It is possible to evaluate a neuromuscular system defect from an adequate history and a meaningful examination. Unfortunately, as has been stated, objective signs are rare and most examinations elicit the manifestations of subjective symptomatology. The complaints are often unrelated to the degree of soft tissue injury.

Muscular pain, albeit with objective intrinsic pathological changes, is also accentuated by the concurrent emotional and thus psychological aspects. Muscular symptoms are also a manifestation of a neural component to the insult. To merely treat the muscular aspect with modalities such as massage, local heat or ice, ultrasound, and exercise is justified if carried on for a limited period of time with explanation and self-administration. The outcome of such treatment usually remains subjective and palliative, thus it tends to be extended beyond reason.

The plea remains that a meaningful evaluation be undertaken and the resultant therapeutic administration be appropriate for the condition and be given for a reasonable period, with attempts being made to document as objectively as is currently possible the results of treatment.<sup>75,76</sup>

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## CHAPTER 5

# Lumbar Discogenic Pain

It has been suggested that the mechanical aspect of low back pain syndromes be divided into discogenic or myogenic etiology.<sup>1</sup> The differentiation is actually whether low back pain is exclusively in the low back or associated with leg pain. The latter implies that the nerve root is involved and that the most probable cause is disk herniation into the foraminal space.

Here, again, objective signs are mandatory yet limited and pain is the predominant sign that implicates the pathology and the involved tissue. Here, again, subjective pain is the presenting complaint and objective signs are used to indicate the impairment that allegedly causes the pain. The objective signs are essentially the signs of nerve root involvement, with low back pain signs relatively less objective.

Pain, again, is the basis for intervention, conservative or objective, with removal or diminution of the objective losses, sensory or motor, incurred being secondary. Only when there is caudal equinal syndrome findings is surgery urgent or even mandated.

## DISK DISEASE

The term *disk disease* has emerged as if there is such an entity as defined. *Disease*<sup>2</sup> from the French *des plus aise* (ease) means literally “the lack of ease,” a pathological condition of the body that presents a group of clinical signs, symptoms, and laboratory findings peculiar to it, differing from other normal or pathological body states. The major difference between disease and illness is that the former is usually tangible and may be measured, whereas illness is highly individual and personal (subjective disability versus impairment).

Pain, suffering, and distress are synonymous with illness and not necessarily with disease. The term *dyscrasia*—means “illness” yet with no evidence of disease as measurable by pathological changes in the body.

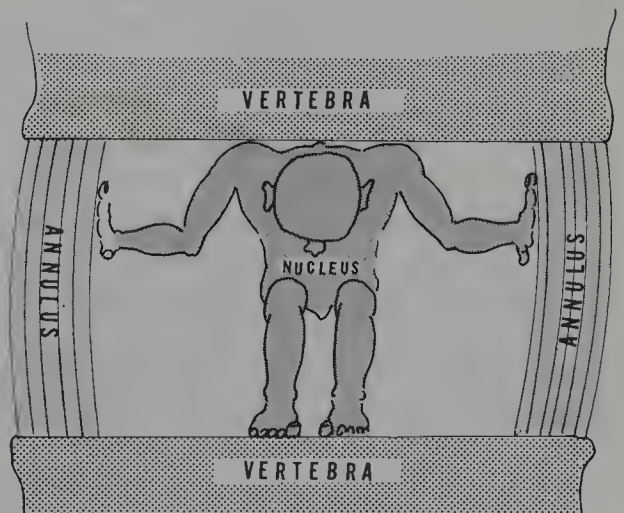
Disk disease has veritably become a specific organic disease entity yet does not enjoy unanimous understanding of its etiology, mechanisms, or even its toxonomy, which is evident with the array of terms, such as *bulging*, *herniation*, *rupture*, *slipping*, *degeneration*, *extrusion*, and so on.<sup>3</sup> Most commonly used, the term *herniation* becomes more meaningful when defined as an abnormal rupture or protrusion of an organ or other body part through an abnormal or natural opening in the wall of the containing cavity.

The pathological changes in the normal disk that ultimately cause symptoms have been reasonably well documented. [The annulus normally resists the internal pressure of the nucleus in its function as a hydrodynamic tissue entity.] The nucleus is a self-contained hydrophilic tissue that separates the vertebral endplates (Fig. 5-1) yet permits compression by deformation and sagittal, lateral, and rotatory motion as permitted by the surrounding annular fibers (Fig. 5-2). The nucleus also exerts lateral pressure outward against the constraints of the surrounding annular sheets (Fig. 5-3).

The annular fibers are the key to the integrity of the intervertebral disk. The layers of the annulus are composed of parallel collagen fibers (Fig. 5-4). Each fiber has a precise degree of elongation that fails when exceeded.<sup>3</sup>

Within the annulus the fibers are angled: originating from the endplate of the superior vertebrae to the endplate of the adjacent inferior

Figure 5-1. Schematic portrayal of nucleus functions. The concept of a force separating the two adjacent vertebrae as well as exerting outward force on the annular fibers is depicted. (From Cailliet, R: *Understand Your Backache*, FA Davis, Philadelphia, 1984, p 17, with permission.)





**Figure 5-2.** Annulus fibrosus. The annulus fibrosus of the intervertebral disk comprises sheets of parallel annular collagen fibers that run obliquely in each sheet and in different directions in each subsequent sheet. The annulus contains the central nucleus not comprised of sheets.

vertebrae (Fig. 5-5). They have a different length, hence a different physiological elongation dependent upon their location within the disk (Fig. 5-6).

Compression of the disk, which is the major function of the disk, merely changes the angulation of the annular fibers (Fig. 5-7) but does not exceed their elongation potential.

Excessive compression has been shown to break the adjacent endplates before causing any tearing of the annular fibers (Fig. 5-8), whereas rotational forces are known to cause tearing of the fibers. Shear and torque forces have been mechanically defined, as have the changes within the annular fibers in compression flexion and extension (Fig. 5-9). The former are potentially destructive, whereas the latter are physiologically needed in the nutrition of the disk via imbibition.

The mechanism by which the disk (annulus) is torn is elicited from the history. Knowledge of the extent of the disk herniation of annular fibers remains for in vitro studies and to a degree from computerized axial tomography (CAT) and magnetic resonance imaging (MRI) studies.

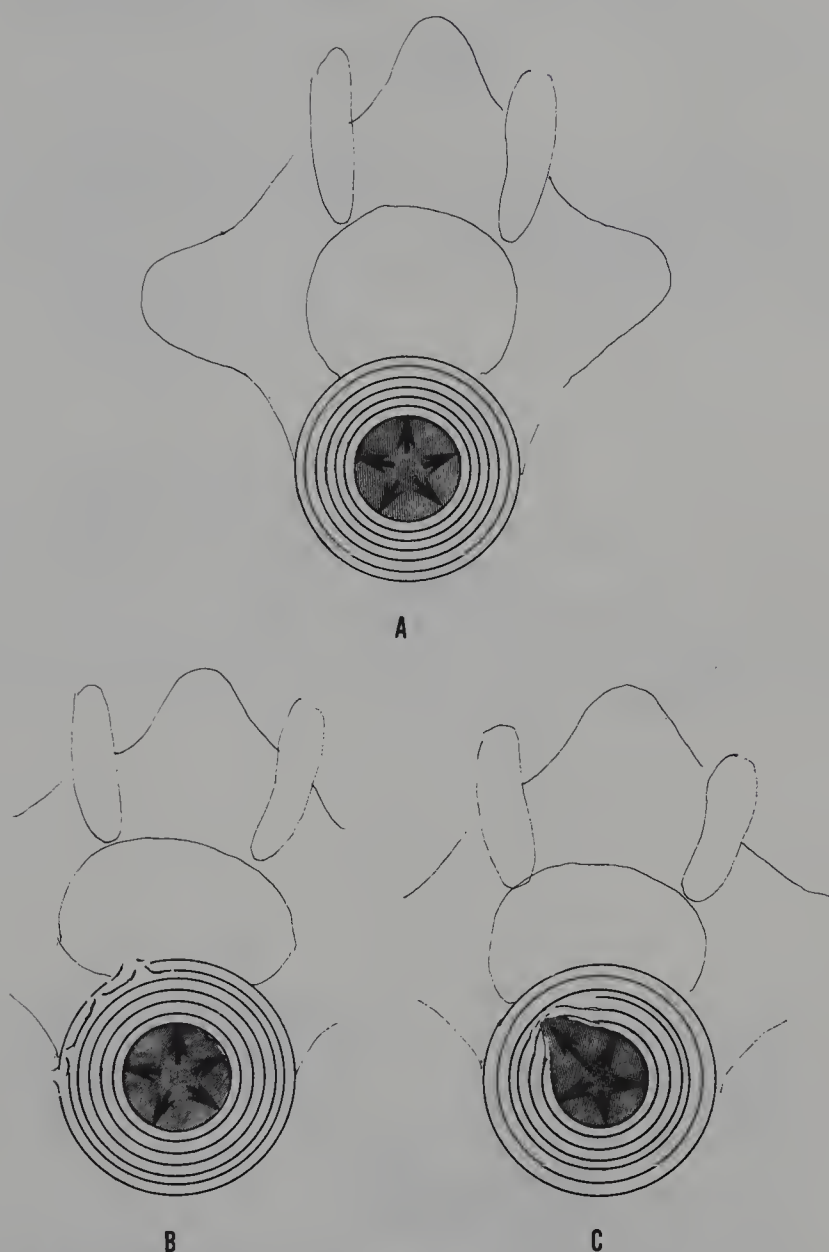
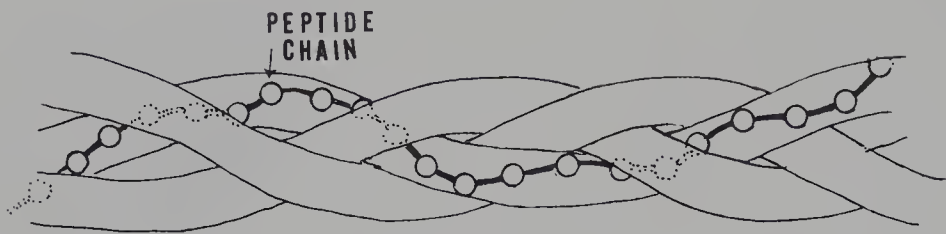
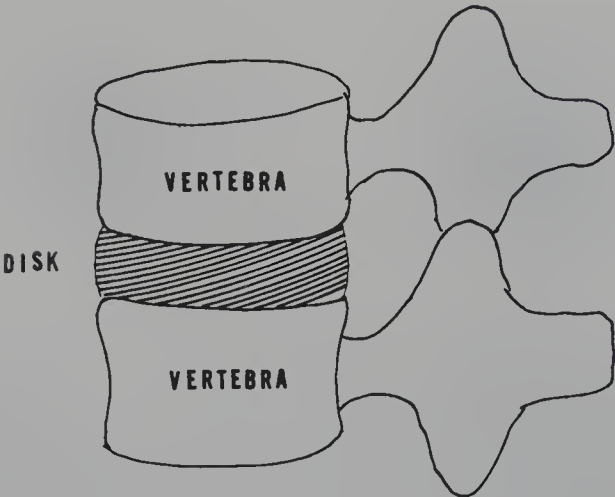
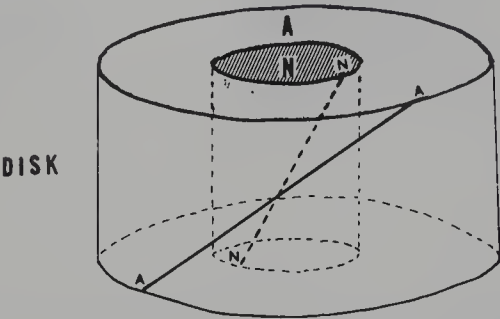


Figure 5-3. Intact disk and nucleus with defects. *Panel A* depicts the intact disk with intact annular fibers and generalized forces in all directions from the nucleus. *Panel B* shows tears in the peripheral annulus that does not disrupt the nuclear forces. *Panel C* shows internal annular fiber tears that allow the nucleus to internally herniate yet the outer annular fibers remain intact.





**Figure 5-4.** Collagen fiber. The collagen molecule is a trihelix peptide chain with each chain containing amino acids. There are in this type-I molecule two alpha-1 and one alpha-2 peptides with each third molecule an amino acid. (Modified from Alberts, B, et al: *Molecular Biology of the Cell*. Garland. New York, 1983, p 694.)



**Figure 5-5.** Annular fibers. In the annulus there are variations in the angulation within each adjacent sheet. The angle of the outer fibers (A-A) is approximately 30 degrees. At the inner layers, near the nucleus (N-N), the angle is more acute and the length is shorter.

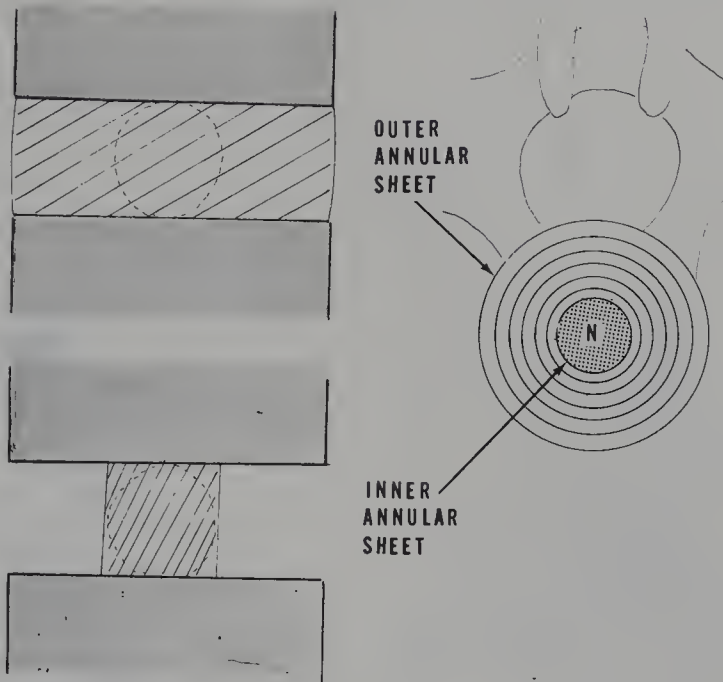


Figure 5-6. Angulation of the annular fibers. The upper left figure depicts the angulation of the annular fibers in the outer layers of the disk. The lower left figure depicts the more acute angle and the shorter length of the fibers.

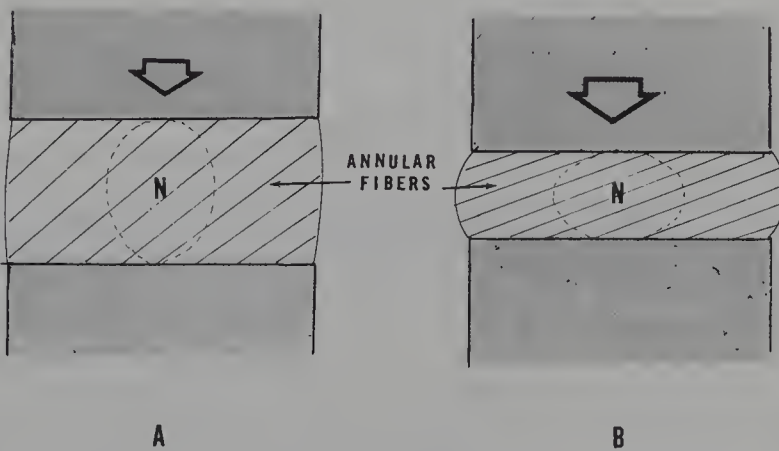


Figure 5-7. Change in annular fiber angulation from compression. The angle of the annular fibers with disk at neutral state (A). When the disk is compressed the angulation of the fibers is greater. N depicts the nucleus, which deforms when compressed.

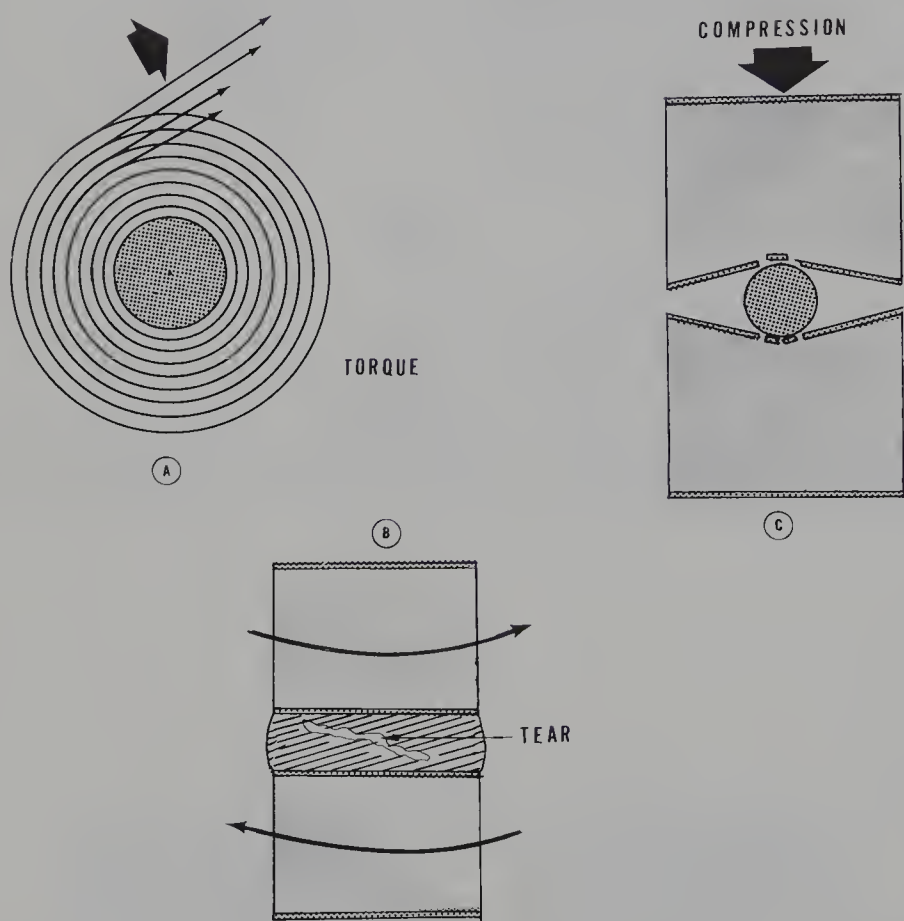


Figure 5-8. Compression compared with the torque effect on the annulus. *Panel A* demonstrates the force of torque on the annulus. *Panel B* depicts tearing of the annual fibers from torque force. *Panel C* demonstrates that when there is severe compression the endplate often disrupts sooner than does the disk annulus.

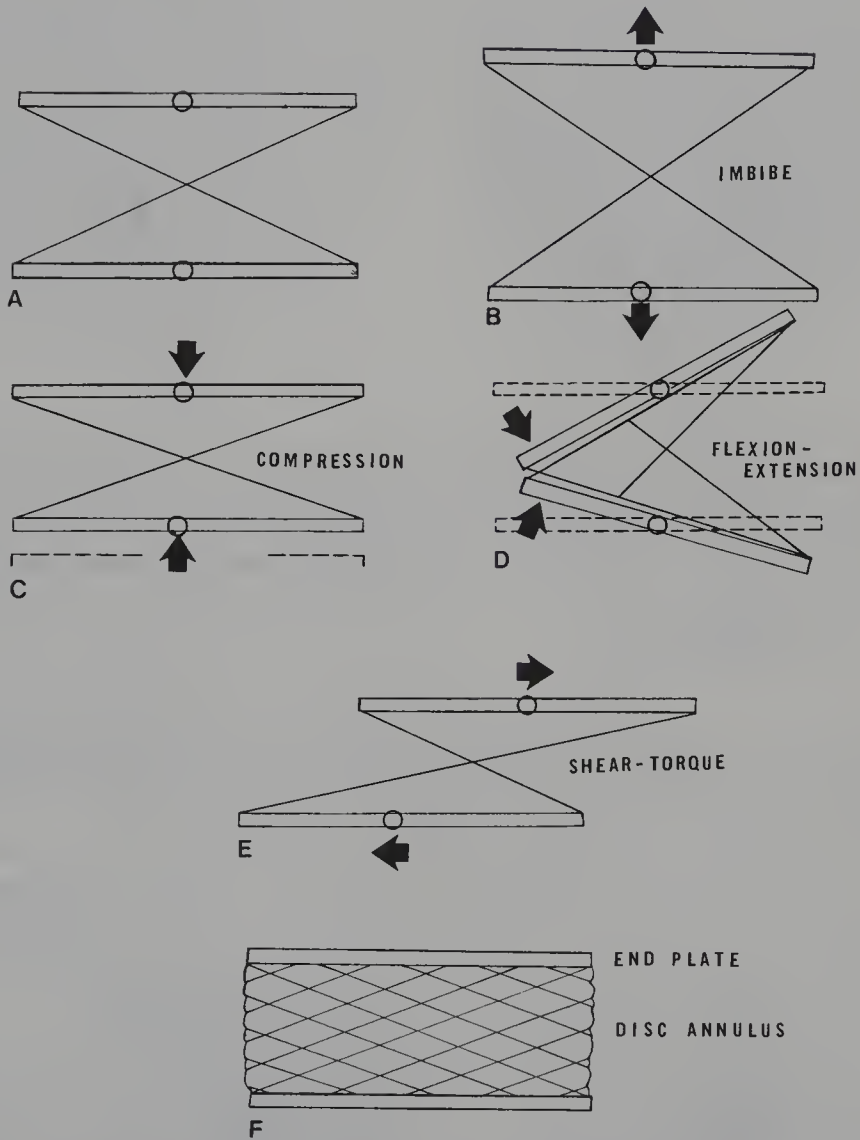


Figure 5-9. Schematic effect of compression, flexion-extension, and shear-torque on collagen fibers. The angulation of the disk annular fibers at rest (A). When the disk expands to imbibe fluids, the angulation changes (B). When compressed the angulation becomes more acute (C). In flexion-extension the fibers change angulation, depending on their position near concavity and convexity (D). When shear-torque forces are translatory a marked elongation of the fibers occurs, often leading to disruption (E). The annular fibers attach to the adjacent endplates of the vertebrae and their angulation (F).

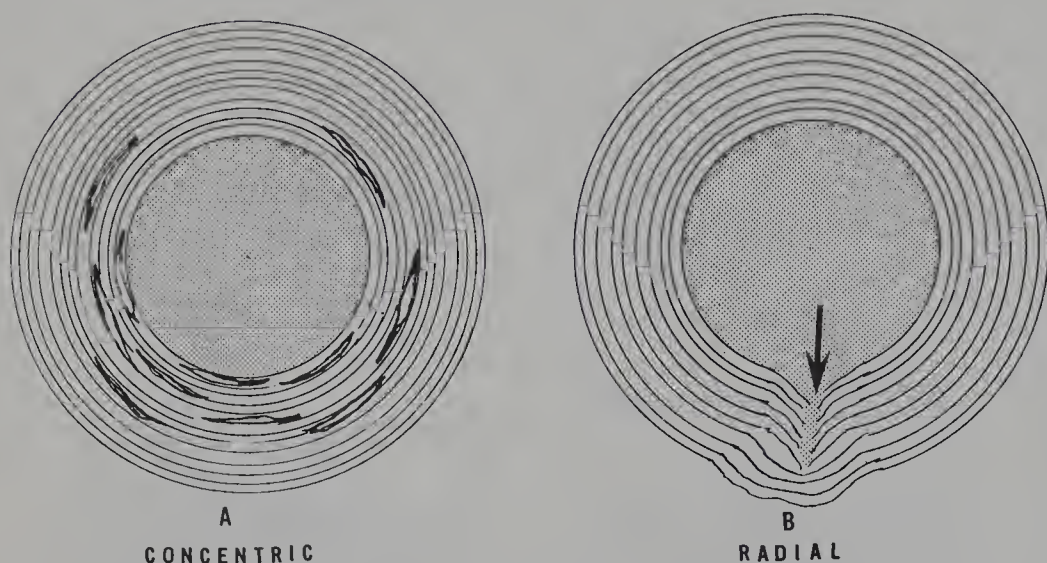


Figure 5-10. Concentric and radial tears of the annulus. Concentric tearing of the annular fibers (A). These tears are essentially separation of the layers from each other, causing a weakness in their integrity. When inner fibers tear they permit the forces within the nucleus to herniate externally causing radial tearing (B).

The original findings of annular tearing were by Hirsch et al.,<sup>4</sup> who found early concentric tears (Fig. 5-10) especially in the posterolateral aspects of the disk. These tears ultimately became radial.

Symptomatology occurs when the internal fibers tear ("internal" herniation of Crock<sup>5</sup>), allowing the nucleus to cause the overlying annular fibers to bulge (Fig. 5-11). If that bulge presses centrally against the posterior longitudinal ligament, low back pain occurs. If it bulges posterolaterally against the nerve root, radiculopathy occurs.

Only when the overlying annular fibers completely tear does the nucleus extrude (Fig. 5-12). This differentiation in terms is very significant diagnostically yet difficult to ascertain clinically or radiologically.

Recent studies have shown that discrete peripheral tears within the annulus lead to secondary changes within the disk and ultimate degeneration.<sup>6,7</sup> Shear forces have also been incriminated (Fig. 5-13).

## CLINICAL MANIFESTATION

The diagnosis of herniated disk as a cause of pain cannot be made on imaging tests alone in the absence of a clinical picture.<sup>1</sup> The clinical picture, however, presents few specific objective signs unless there is also radicular pain.



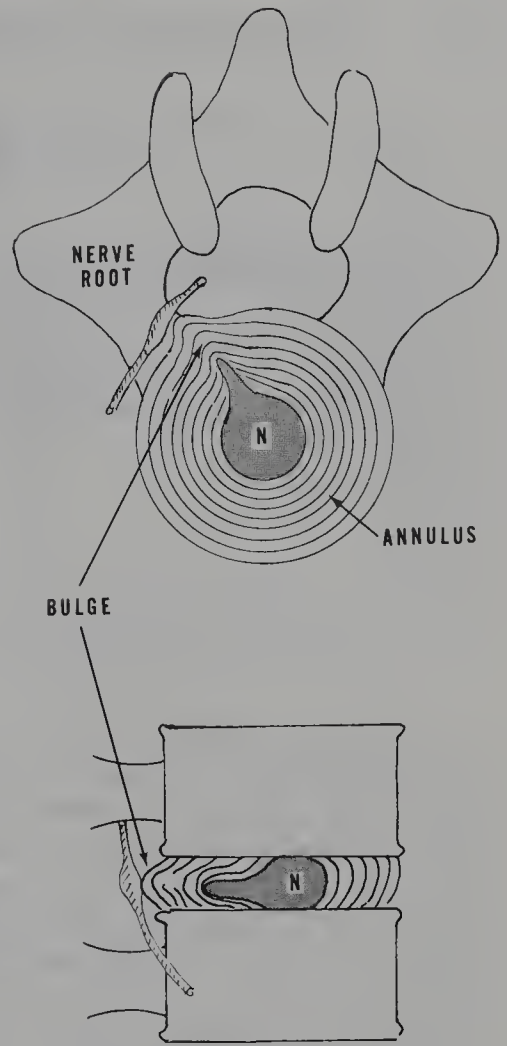


Figure 5-11. Central disk herniation with annular bulge. The nucleus herniating externally through inner annular tears, forcing the outer annular fibers to bulge into the intervertebral foramen toward the nerve root (*top*). A lateral view of this event (*bottom*).

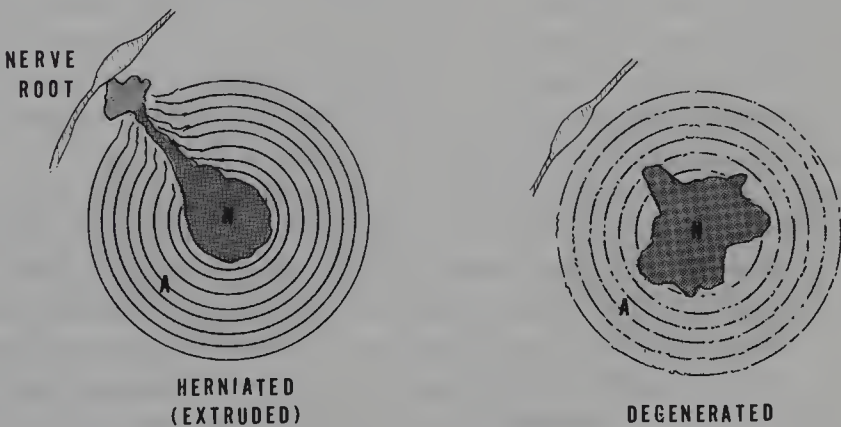


Figure 5-12. Extrusion of the nucleus. Notice how the nucleus extrudes from the disk through the entire annular tear. In precise terminology that is truly a *herniated nucleus* or an *extruded nucleus* (*left*). Notice how internal extrusion within an otherwise normal annulus causes the nucleus and inner annulus to degenerate (*right*).

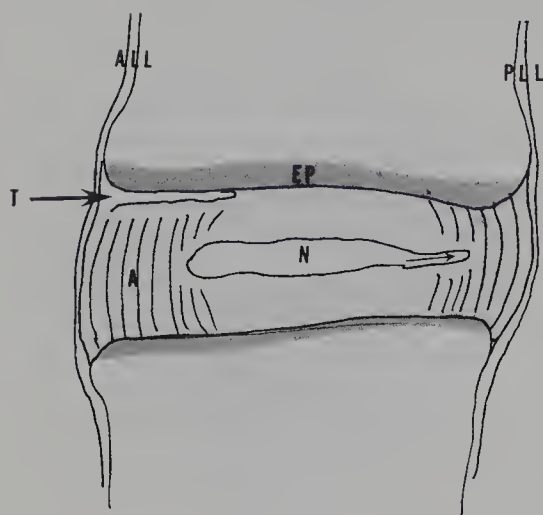


Figure 5-13. Annulus tears and intervertebral disk degeneration. A degenerated disk, showing the nucleus (N) being deformed. A forceful translatory force can tear (T) the outer annular fibers (A) from the vertebral endplate (EP). The anterior longitudinal ligament (ALL) can also be torn from its vertebral attachment. The more distal posterior longitudinal ligament (PLL) does not become involved. This type of disk injury occurs usually from a severe external translatory force.

Annular tear allows mechanical pressure upon sensitive adjacent tissues.<sup>8,9</sup> These are the posterior longitudinal ligament, the nerve roots, and their dura. Back pain alone is manifested by pain on trunk flexion and limited range of motion. *All* subjective signs are then considered due to disk herniation if there is radiological evidence. The literature is rampant with studies in which low back pain occurs without central disk herniation and where disk herniation occurs without pain and limitation.

Most low back pain is attributed to different problems.<sup>10</sup> If they are all added up, they account for 200% to 300% of persons with back pain.<sup>11</sup> Of five treatments advocated, all claimed 80% success rates,<sup>10</sup> implying that no matter how patients are treated, 80% improve. The costs of diagnosing and treating spinal disorders have been estimated (see Table 5-1). From Table 5-1 and all the entities involved in the care, it is obvious that there is a strong financial appeal to evaluate and treat the common low back pain.

In 1987 a report entitled "Scientific Approach to the Assessment of Activity-Related Spinal Disorders" was published in *Spine*.<sup>12</sup> This profound and provocative report was prepared by the Quebec Task Force (QTF), which had been commissioned by the Quebec Institute for Workers Safety and Health to report on the current status of diagnosis and treatment of activity-related spinal disorders. This study was initiated by the rapid escalation of the cost of the therapies related to the numerous complaints of low back pain as a disability.

The QTF stated, "Of the numerous pathological conditions of the spine, nonspecific ailments of back pain in the lumbar, dorsal and cervical regions, with or without radiation of pain, comprise the vast majority of problems found among workers." Similar problems occur among

Table 5-1. ESTIMATED DIRECT COSTS OF SPINAL DISORDERS

<i>Charges</i>	<i>Cost (\$)</i>
Hospital inpatient	6,780,462
Outpatient and emergency room	387,980
Outpatient diagnostic and therapeutic	2,000,000
Physician inpatient	1,707,080
Physician outpatient and emergency room	2,411,690
Other practitioner	2,825,119
Drugs	191,697
Nonhealth goods and services	1,564,651
Total	17,868,679

Modified from Cats-Baril, W, and Frymoyer, JW: The economic impact of spinal disorders. In Frymoyer, JW (ed): The Adult Spine. Raven Press, New York, 1991.

nonwork-related injuries and constitute complaints after personal injury.

It is estimated that 90% of spinal mechanical disorders fall into this category.<sup>13</sup> Thus only 10% of these patients can be objectively diagnosed and thus objectively treated. The QFT recommended that "pain patterns be used as the basis for . . . a universal classification." Again, the lack of objectivity and the existence of subjective pain complaint only becomes the norm. The basis of this text is evident.

The following are paraphrasings of the QFT classifications.

1. Pain in the lumbar, dorsal, and cervical area. "The pain is intermittent or constant, its intensity varying with the patient's tolerance, and is almost always aggravated by mechanical factors." (There is no mention of emotional or psychological factors.)
2. Pain in the lumbar, dorsal, or cervical areas with radiation proximally (but not beyond the knee) and not accompanied by neurological signs. This pain can be neurogenic but originates from deep structures of the rachis.<sup>14,15</sup>
3. Pain with radiation (beyond the knee) without neurological signs may suggest radicular origin or be diffuse. (Again, this is a vague subjective sign without objectively.)
4. Pain with presence of neurological signs may result from various affections, the most frequent being diskal herniation. (The relationship, however, of objective findings with subjective complaints remains obscure.)

This astute research study clearly defines neurological signs as the only objective sign and every other aspect a subjective claim of disability and impairment. Even with the presence of objective neurological signs, their relationship to subjective symptoms is not clear.

McKenzie amplified these categories by concentrating on the distribution of the pain: the pattern of the QTF report.<sup>13</sup> He classified pain as “central” and “distal,” with the objective of the treatment being to change the pain pattern from distal to central. His concept related to the extent, site, and distribution of the referred leg pain. With the etiology being considered as nerve pressure from a disk protrusion (Fig. 5–14), the concept is valid.

The dermatomal patterns or radiculitis commonly featured in neurological texts that have been discussed in Chapter 4 are not that precisely identified by patients nor that clearly elicited by the clinician, but they suffice to indicate referred pain above or below the knee. This differentiation is mandatory because the sciatic nerve innervating the area below the knee emanates from the foramina between L4, L5, and S1 (Fig. 5–15).

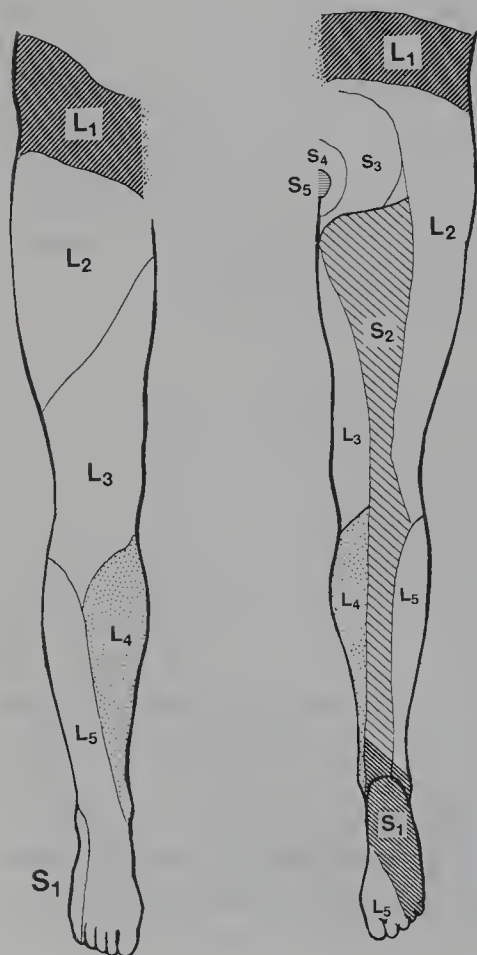
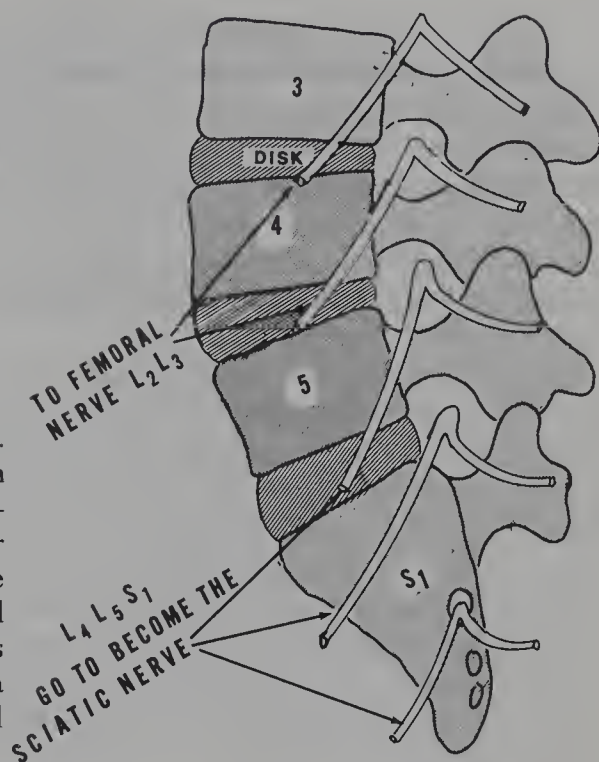


Figure 5–14. Subjective area of referred pain. The classic dermatomal areas of referred pain are depicted.



Figure 5-15. Sciatic nerve roots. The nerve roots that emerge from the intervertebral foramina between the fourth and fifth lumbar and the sacrum are the nerve roots L4, L5, and S1 that descend to form the sciatic nerve. Roots emerging from higher foramina (L2 and L3) form the femoral nerve.



The McKenzie concept implies that the degree of annular deficiency determines the degree of referred pain.<sup>16-18</sup> As the annulus sheets tear in successive layers, the space between these involved layers becomes occupied by fluid gel or sequestrum. The volume of protruding disk thereupon increases, with a resultant increase in the referred site of pain. If the insults to the disk continue or are repeated, the remaining (intact) layers of the annulus may fail, ultimately allowing extrusion of the nucleus (see Fig. 5-13). The material extruded is no longer reducible.

This concept has been termed the *derangement syndrome*,<sup>13</sup> with the implication that repeated or persistent motions that cause migration of the nucleus posteriorly cause progressive tearing of annular sheets. These offending motions are motions of lumbosacral flexion.

Usually before the nucleus extrudes or the material between the torn annular sheets becomes fibrotic, scarred, contracted, or adherent, the internally extruded nuclear material can be mechanically returned to a more central position. The pain allegedly also migrates centrally from its distal site as improvement occurs.

This approach conceptually differs from the traditional Williams flexion exercises that have been advocated for both benign low back pain and herniated lumbar disk.<sup>19-22</sup> Clinical outcome assessments from this procedure have been favorable.



The clinical determination as to the therapeutic protocol is the production of the pain and its distribution from specific maneuvers and positions that are subjective; however, the objective neurological signs, including the straight leg raising (SLR) test, have been documented. The therapeutic protocol of this concept will be discussed in Chapter 10.

The algorithm problem-solving approach has been strongly endorsed by many prominent clinicians, but most of the sequences are subjective.<sup>23</sup>

The decision for surgical intervention relies primarily in the proper, precise diagnosis. This is a difficult decision, as the precise tissue site of nociception that impairs or disables the patient is not always clear<sup>24</sup>; therefore, its surgical removal is not always predictable. Only “when neurological (objective) deterioration is ‘increasing’ is it ethical to proceed with imaging studies with the anticipation that surgery might be necessary in the subacute time span.”<sup>24</sup>

The presence of radiological abnormalities alone or the mere presence of pain is not a valid basis for surgical intervention, although “radiological confirmation of pathology” has become mandatory in most clinicians’ decisions. “Surgical care for discogenic pain is unwarranted.”<sup>24</sup>

As will be discussed in Chapter 7, there have been subsequent studies to document “quantification of function.”<sup>25</sup> These have been designated into several phases:

1. Acute phase (from date of injury or postoperative), being essentially 8 weeks
2. Subacute phase, extending from 6 weeks to 4 months
3. Chronic phase, involving 4 months

How these dates have been designated is obscure and actually controversial. Many of the functional tests,<sup>26</sup> such as range of motion, strength testing, and functional capacity, have been discussed, but most are based on instrumentation analysis, which currently defies objectivity and reproducibility. None necessarily simulates the exact factors of the daily function it is testing, including the fatigue, ergonomics, frame of mind, or job satisfaction.

This testing procedure also applies in the evaluation of functional capacity testing in the consideration of a work hardening protocol. In this procedure the testing therapist “must review medical records and test for flexibility, strength and symptoms.”<sup>26</sup> All have been discussed and found wanting.

In discussing objective findings termed *structural diagnostic testing*,<sup>27</sup> White succinctly summarizes their value as paraphrased by the author:

1. Imaging confirms; it does not diagnose.
2. Gadolinium enhances MRI and differentiates scar from disk herniation after failed surgery.
3. Scans do not indicate where the pain is coming from.
4. An MRI may be negative yet the discogram may be abnormal. (The latter, however, may also not be precisely diagnostic.)
5. CAT scans and MRIs miss early internal disk disruptions, early facet degeneration, spondylolysis, arachnoiditis, and soft tissue lesions.

Studies have shown that 90% of patients operated on have returned to work in 4 years,<sup>28</sup> yet 92% have returned to work after conservative (nonoperative) treatment.<sup>28,29</sup> These comparisons are not very meaningful, as most diagnoses and outcomes were largely subjective.

It may well be summarized that the evaluation (diagnosis) of the patient with low back *and* related leg pain demands that only the objective signs, meaning a properly evaluated SLR test and objective neurological examination, be documented and reconfirmed over a significant period of time. Pain still remains the major cause of disability and eludes objective documentation.

## PHYSICAL EXAMINATION: OBJECTIVE FINDINGS

The objective findings on physical examination to confirm the pathology documenting impairment are scarce, as has been stated. The SLR test (Lasègue test), admittedly an admixture of objective and subjective components, has been considered an objective test to implicate nerve irritation from encroachment upon the nerve root in the paraspinous area, the dorsal root ganglion or the nerve root anywhere in the foramen. This test is primarily a confirmatory test for leg pain complicating low back pain and incriminating the intervertebral disk. This test has been intensively discussed in Chapter 3 and will not be reiterated here except to highlight aspects relating it to the intervertebral disk.

### Straight Leg Raising Test

The positive SLR test has been accepted as a valid objective clinical test to demonstrate an inflammatory compressive process upon the precise nerve root within a foraminal canal. It has been used clinically to confirm a herniated lumbar disk.

Forst first advocated the SLR test in 1881 and advocated raising the fully extended leg.<sup>30</sup> Lasèque discussed the subject of sciatica in 1864 but did not describe the test that now bears his name.<sup>31</sup> It is interesting that the original illustration of the test had the patient with his neck flexed but did not discuss this aspect. Both Forst and Lasèque attributed the resultant pain as resulting from stretch of the inflamed posterior thigh muscles (hamstrings) and not the sciatic nerve.

Fajersztajn further explained the mechanism of a positive SLR by adding nuchal flexion and ankle dorsiflexion.<sup>32</sup> (See illustrations in Chapter 3.) The mechanism ultimately was resolved as the pain coming from the dura and not the nerve itself. In the Fajersztajn test it is the dura that becomes stretched and not the nerve. The crossed (contralateral) sciatic SLR test, where ipsilateral sciatic pain is elicited by contralateral SLR implicates the dura. Charnley in 1951 termed the SLR test as more important "than all the other clinical or radiological signs put together."<sup>33</sup> This statement is true *if* the test is properly applied and clinically interpreted.<sup>34</sup>

The SLR test implies that the nerve root moves within the canal,<sup>33-37</sup> but initially movement was considered in a distal migration: along the direction of the emerging nerve from the cord. Increased nerve root tension from an external source (herniated nucleus pulposus [HNP] or osteophytes) was considered the cause of radicular pain.<sup>38</sup>

The displacement (movement) of the nerve roots and their direction were never completely evaluated until a recent excellent study to determine precise extent of movement, direction, and the effect of fusion of a functional unit upon these motions.<sup>39</sup> These results revealed movement of 1.4 mm (at L4 space), 2.1 mm (at L5) and 2.5 mm (at S1). Movement occurred distally (parallel to the nerve) but also laterally upon gradual SLR. This finding confirms the factor of pain at certain degrees of SLR and the difference in lateral disk herniations.<sup>40</sup>

This factor also explains a positive SLR in degenerative disk disease (without herniation) with nerve entrapment in the lateral recess<sup>41</sup> (Fig. 5-16).

The relationship of the nerve root and its dura also has not been evaluated completely. The dura is described as an inelastic structure that is relatively lax and made more tense by the cerebrospinal fluid (CSF).<sup>38</sup> This may explain how the Valsalva reaction is a sciatica from HNP.

In SLR the dura moves less than does the enclosed nerve root.<sup>39</sup> At the axilla of the dural sleeve the dura is relatively fixed (Fig. 5-17). Tension within the dural sac undergoes the principal of Saint-Venant's law (Fig. 5-18). The nerve root tends to move against the superolateral edge of the dura. As the dura is relatively fixed at the pedicle and more mobile at the level of the dorsal root ganglion, the dura experiences

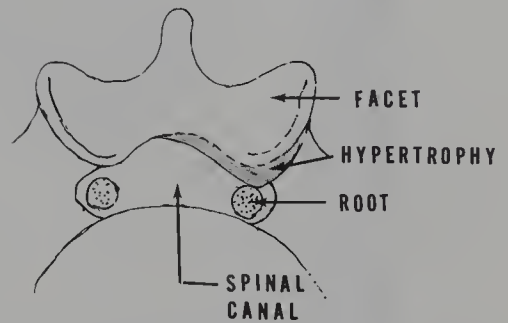
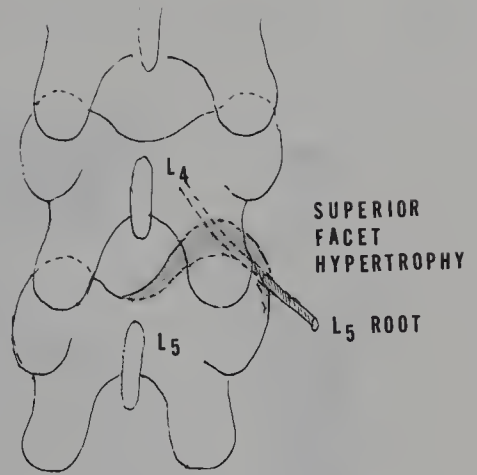


Figure 5-16. Foramenal stenosis and root entrapment. In this illustration the foramen is stenosed by virtue of a hypertrophic superior facet entrapping the L5 root. The *upper view* is from behind and the *lower view* is from above.

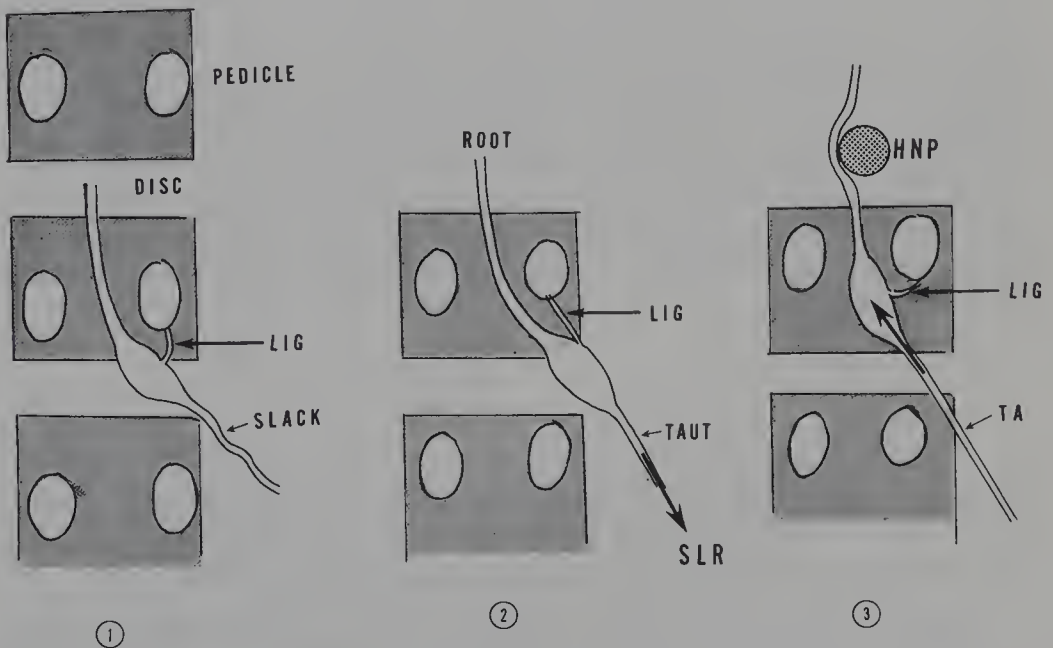


Figure 5-17. Nerve root traction. The nerve root is slack (1). The effect of traction on the nerve root during straight leg raising (SLR); downward migration of the root is prevented by the ligament (LIG) that causes increase in nerve traction (2). A herniated disk (HNP) on the nerve root pulls the root cephalad, causing traction (3).



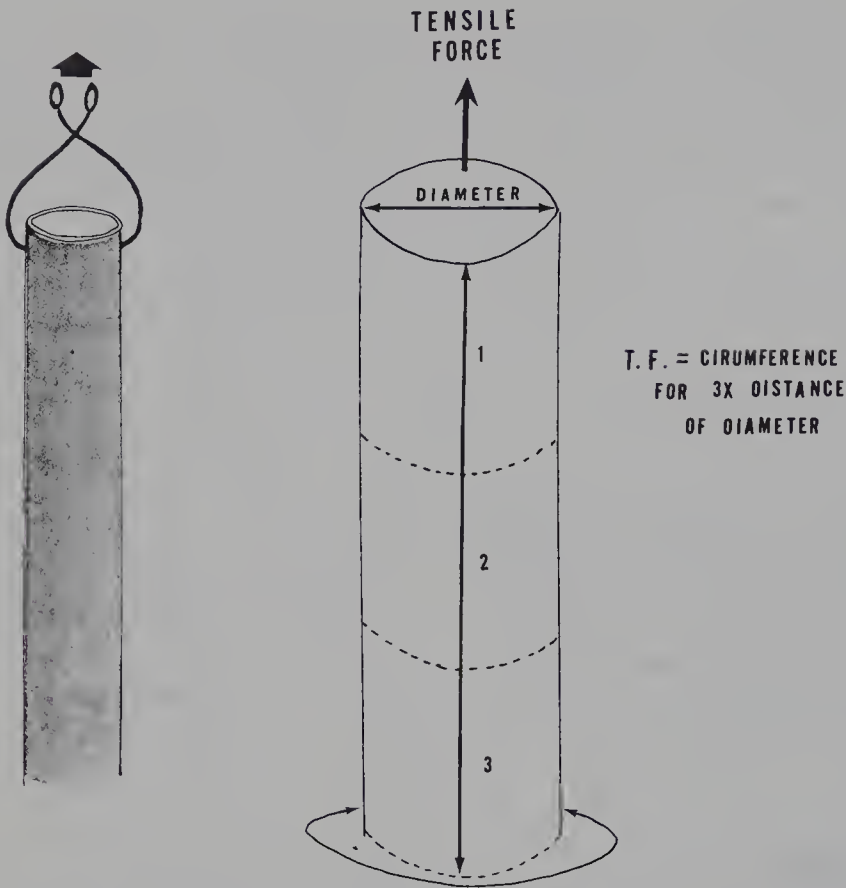


Figure 5-18. Saint-Venant's law. This law states that tensile force applied in a longitudinal direction is distributed uniformly around the circumference for a distance equal to three times the diameter of the tube. Thus tension applied to a nerve root dura (tube) is transmitted as a compression force for a distance of three times the diameter of the root both caudally and cranially. This explains radicular pain being elicited from a compressed root by SLR and nuchal flexion or ankle dorsiflexion.

more strain with SLR than does the nerve root.<sup>42</sup> Both the nerve root and the dura undergo stretch in SLR.<sup>43</sup> Nerves can normally undergo 15% elongation without permanent structural damage.<sup>32,35</sup>

The dura has been proved to have nociceptive innervation from the nerve of Luschka (Fig. 5-19), whereas the nerve root itself has not been shown to have significant innervation.<sup>44</sup>

In spinal stenosis, especially foraminal stenosis (see Fig. 5-16) or tropism of the lumbosacral spine (Fig. 5-20), the nerve root can be entrapped. Evidently, sciatic radiculopathy can occur from degenerative disk disease.



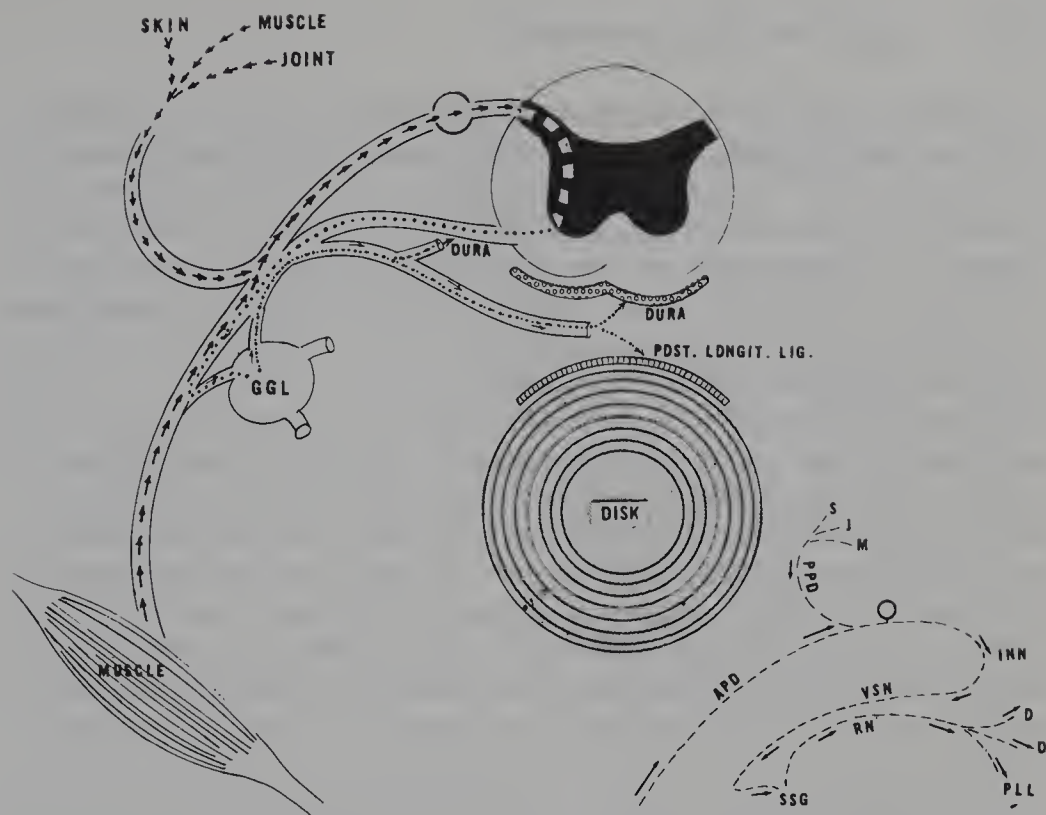


Figure 5-19. Innervation of the recurrent nerve of Luschka. The recurrent nerve of Luschka (RN) supplies the posterior longitudinal ligament (PLL), the nerve root dura (D), and conceivably the very outer layers of the annulus. APD = anterior primary division; GGL = sympathetic ganglion; INN = inter-nuncial neurons; PPL = posterior primary division; SSG = sensory sympathetic ganglion; VSN = ventral sensory nerve.

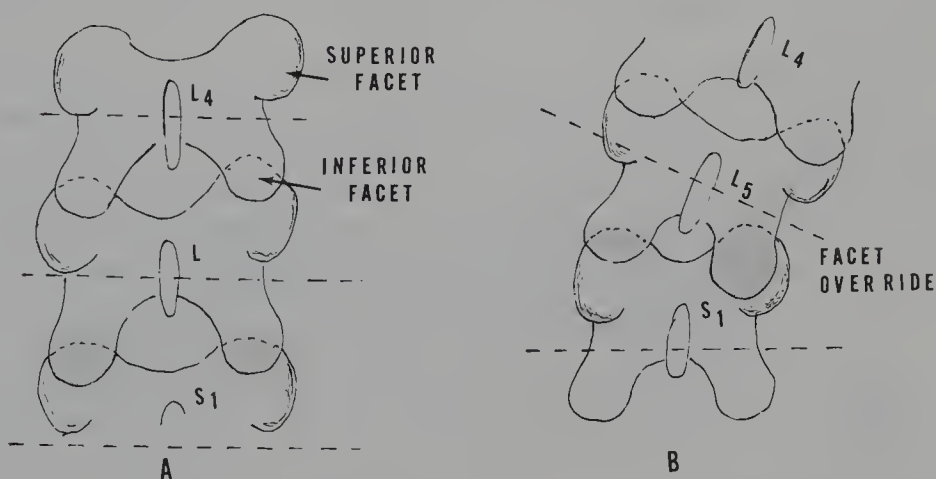


Figure 5-20. Tropism of lumbosacral spine. The term *tropism* has been applied to asymmetry of weight bearing and alignment of the lumbosacral articulations (A). This asymmetry causes an irregular shape of the disk and asymmetry of the facet alignment (B).

The decision to fuse the segment after discectomy to prevent instability was initially advocated. Instability was described as "the loss of the ability of the spine under physiological loads to maintain the relationship between vertebrae in such a way as there is neither initial damage nor subsequent irritation to the spinal cord or nerve roots."<sup>45</sup> How fusion affects nerve root motion was highlighted,<sup>38</sup> revealing that nerve roots migrate slightly more during SLR after fusion. Thus, fusion does not guarantee relief from sciatica.<sup>46</sup>

A point that needs emphasis is that sciatica from intervertebral constriction must involve the lower nerve roots (L-4, L-5, S-1) as entrapment of higher roots (L-2, L-3) descends the femoral nerve (see Fig. 5-15) and thus is not truly sciatica, nor is it elicited by the SLR test.

The sensory examination determining change in the dermatomes is also subjective but properly administered can have objectivity. The dermatomes have been specified as being consistent in precise dermatomal patterns<sup>46</sup> (with illustration in Chapter 3) but less precise in recent studies. Reference is made to Chapter 3 rather than a repetition here.

Their testing varies with the clinician from pin, light finger touch, cotton application, to pinprick wheel. The technique varies from pinpoint touch to gentle scrape of the testing material. Any are acceptable if done carefully, gently, and precisely. The areas of the dermatome need to be established and described accurately in the chart. The usual designation is "absent or present." To be "diminished" is unclear, as it denotes objective measurement of appreciation on the part of the patient which cannot be quantified. The size of the dermatome found to be affected could be measured so that increase or diminution could be appreciated to evaluate treatment, but this has not been studied or documented.

Unphysiological dermatomal testing results, such as hypalgesia or hypaesthesia in a "stocking distribution," has had a firm acceptance as being in the category of malingering. This is not totally accepted, as the unphysiological area described by the patient merely states that the objectivity of the finding cannot be construed as valid. The motivation is not in question but must be considered in viewing the total picture of the patient's complaints.

Perianal sensory (S2, S3, S4) testing for integrity of the sacral segments is the most valuable objective test as it relates to the possible presence of the caudal equinal syndrome and may not have been noted by the patient.

## Motor Testing

Strength has also been classified as objective testing. For myotomal determination there is more objectivity. The myotomes most frequently

involved in lumbosacral pathology are those to the gastroc-soleus (S1), hamstrings (S1), gluteus maximus (S1), anterior tibialis, big toe extensor and gluteus medius (L5) and quadriceps (L3-4).

Tender spots at the motor sites of the impaired muscles have been claimed to be specific, but these are sensory tests and thus more subjective than objective.

Weakness is also subjective, as it is influenced by effort, avoidance of pain, or inability to isolate function. Testing myotome strength in functional activities such as the gastroc-soleus in toe walking, anticus in heel walking, and quadriceps function in deep knee bends is valid but also not always clear in isolating specific myotomal function.

Fatigue has been utilized by the author in determining myotomal function. This is a subtle sign of impaired function. Repeated extension of the big toe, tested against that of the contralateral side, reveals slight but objective impairment (L5) when fatigue intervenes that cannot be assumed by the patient as it is not functionally impairing. This fatigue testing is also useful in testing ankle dorsiflexion (L4-5). Often this has not been noted by the patient nor is it adequately tested by merely heel walking. The hamstrings (S1) can be tested manually or mechanically (Fig. 5-21) by having the patient in the prone position and having him or her flex the knee. Fatigue can also be elicited. The hamstrings can be tested by resisting the knee flexion with the patient seated as when testing the SLR.

Combining sensory (dermatome) and motor (myotome) testing is the most effective objective testing for confirmation of the accurate diagnosis.

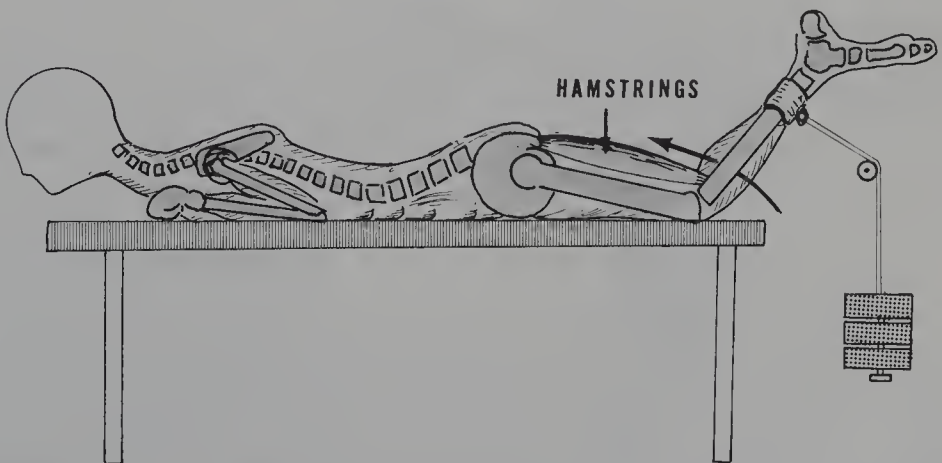


Figure 5-21. Hamstring muscle testing. In the prone position the strength of the hamstring muscles can be tested. This tests the sciatic nerve roots L5, S1, and S2 so that it does not test a precise single nerve root.

## Scoliosis

Acute scoliosis is noted frequently in acute discogenic disease, yet its exact mechanism remains obscure and its diagnostic value unclear. Review of the literature fails to clarify this problem.

Some think that it is a segmental manifestation of posterior primary division nerve involvement causing the ipsilateral muscles to contract (Fig. 5-22). A theory exists that the scoliosis is a reflex mechanism by which the spine flexes away from the nerve entrapment by paraspinous muscle contraction.

Scoliosis caused by a short leg (Fig. 5-23) has been postulated, but its relationship to low back pain or herniated lumbar disk has been refuted.

## Electromyography

Electromyographic confirmation of both sensory and motor impairment adds to the objectivity of the tests. A positive EMG denotes the localization of a precise root and discriminates between acute or chronic lesions, and a negative EMG refutes the presence of a nerve root impairment when properly interpreted. Correlation of the clinical findings, the radiological confirmation, and the EMG constitutes a total judgment, whereas individually none is specifically diagnostic.

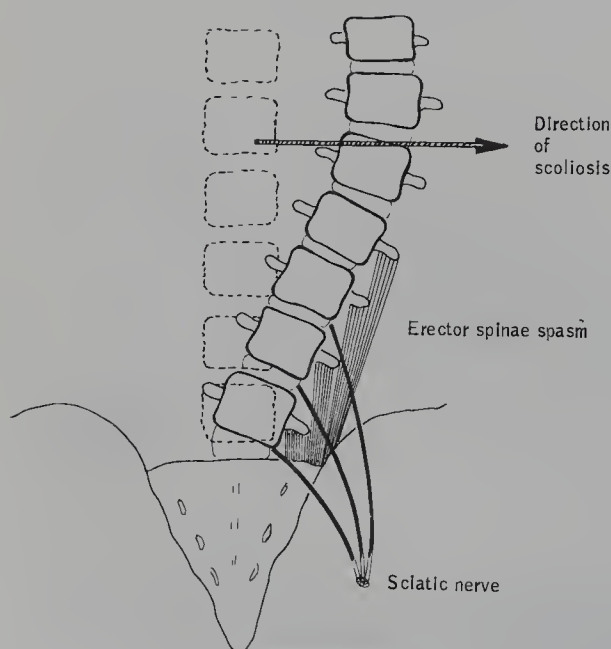
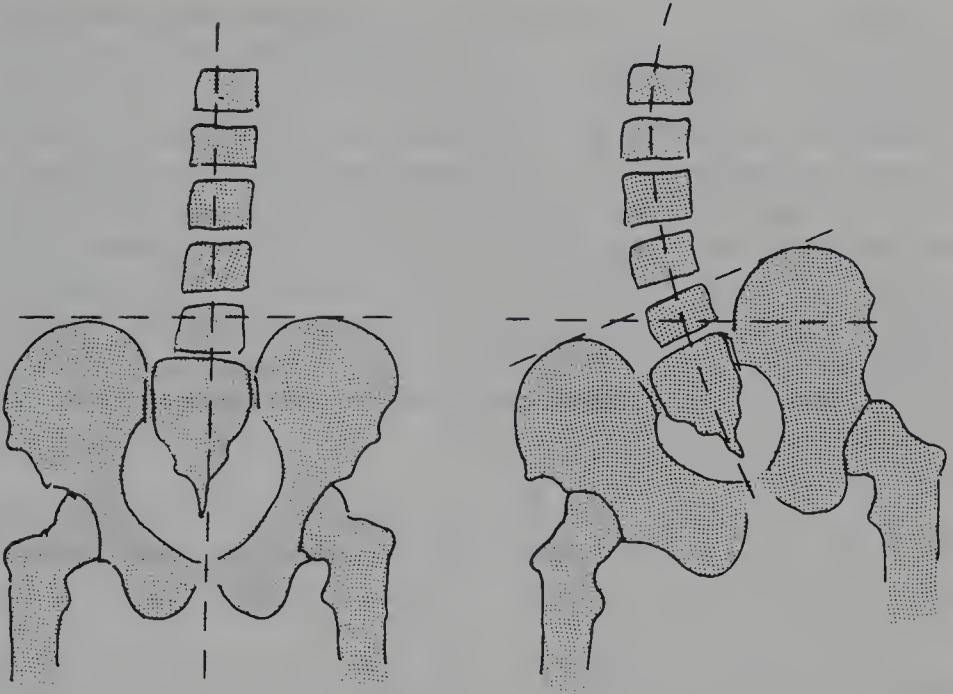


Figure 5-22. Acute scoliosis. The acute scoliosis so frequently noted in a herniated lumbar disk condition has been considered *protective* because the scoliosis is usually away from the sciatic nerve side. This theory has been assumed but not confirmed as the scoliosis often disappears when assuming the prone position.





**Figure 5-23.** Scoliosis due to pelvic obliquity. In the presence of pelvic obliquity, because of a short leg, the superincumbent spine assumes a curvature. This can be ascertained by visually measuring the level of the pelvis and correcting the spinal curvature by placing a measured object under the short leg. (From Cailliet, R: *Scoliosis*. FA Davis, Philadelphia, 1975, p 38, with permission.)

## Upper Motor Neuron Testing

It seems inappropriate to state that upper motor neuronal testing should be included in all examinations, but because they are so often omitted, this must be stressed. A Babinski test is simple, quick, and easily interpreted. No examination should miss this test.

## Confirmatory Studies

Discography has been advocated as a provocative test in which a fine needle has been inserted into the nucleus of the disk and a contrast medium or normal saline injected. Reproduction of the patient's pain has been considered as being positive evidence that the disk is the cause of the pain. This has been true in cervical discogenic disease and also in the lumbar disks.



Analgesic discography is a complement to provocative discography, in which an injected analgesic agent relieves the patient's pain. It is also diagnostic.

Differential diagnosis of low back pain as emanating from the disk, the facets, or both has posed a significant diagnostic dilemma. A provocative study has further added to this dilemma. Although this study was done in the cervical region, there is little doubt that a similar finding would be reached in the lumbar spine. The authors of the study found that 20% of disks alone were positive, zygapophysial joints alone were positive in 23%, 41% found both as pain producing, and 17% found neither to reproduce the local pain. Muscles as the cause of the low back pain were not studied in this report, as they are not reproducible by diagnostic procedures.

False positives were also ambiguous in this study, further casting doubt on the specificity of either diagnostic procedures and in implicating a specific structure as the cause of the low back pain (cervical in this study).

The pathological studies of degenerated intervertebral disks and their sequelae by Vernon-Roberts clearly indicated that degenerative changes are present in all subjects of middle age and are more severe when there is evidence of vertical tears and posterior nucleus prolapse. He concluded that osteophyte formation as well as remodeling (degenerative) changes in the zygapophysial joints accompanies all disk degeneration. The sequence postulated was disk degeneration being the primary event, with the latter being sequelae.

External trauma causing translatory forces has been postulated as being causative of disk degeneration in the cervical spine. It also can be causative in the lumbar spine and lead to all the above sequelae.

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## CHAPTER 6

# The Role of the Facets in Low Back Pain

The zygapophysial (facet) joints have long been considered as being a site and source of low back pain with and without radiculitis.<sup>1,2</sup> In 1911, Goldthwait stated that “the peculiarities of the facet joints” were “responsible for low back pain and instability.”<sup>3</sup> In 1941, Badgley concluded that “the anatomical possibilities of the articular facets to play a more or less active part in the production of low back pain are obvious.”<sup>4</sup>

The term *facet joint syndrome* was added to the toponymy of low back pain.<sup>5</sup> For a while its designation as an etiology of low back pain was diminished but again resurfaced when irritating injections into the joint reproduced low back pain. Injection of a noxious substance into the facet joint caused not only low back pain but also root tension signs and electromyogram changes. Mooney and Robertson found that injecting an anesthetic agent into the facet joint relieved pain for a period of 6 months.<sup>6</sup>

For years numerous articles appeared that implicated the facet joint(s) as a source of low back pain,<sup>7-9</sup> and numerous techniques of treatment<sup>10</sup> emerged, but accurate diagnostic procedures confirming the facets as the site of pain have remained yet unproved.<sup>11</sup>

### STRUCTURE AND FUNCTION OF THE ZYGAPOPHYSIAL JOINTS

The zygapophysial joints are synovial joints between the articular processes of the laminae (Fig. 6-1). These joints with the intervertebral disks form a functional unit.

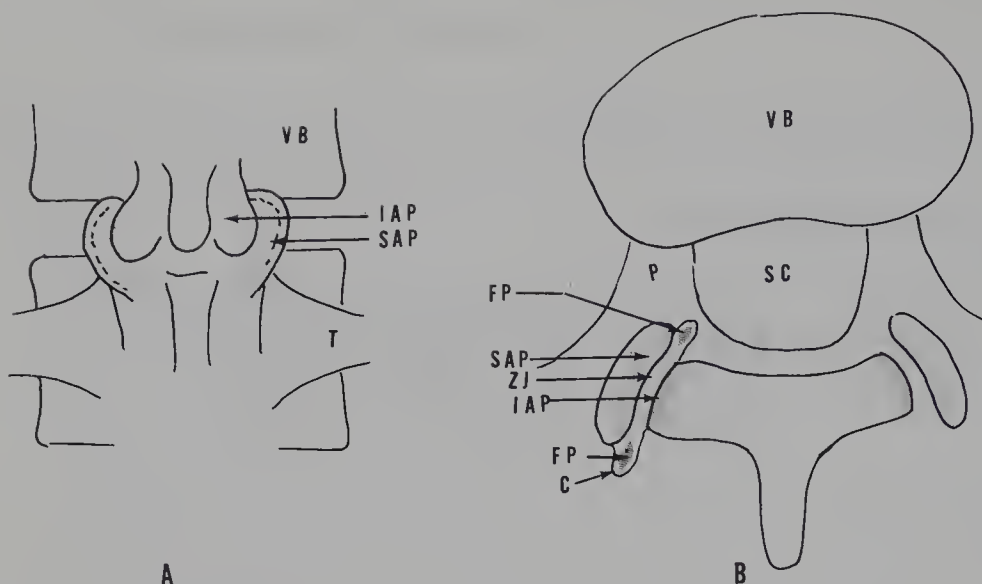


Figure 6-1. The zygapophysial joints (facets). *Panel A* is a posterior view of two vertebrae (a functional unit). IAP designates the inferior articular process and SAP designates the superior articular process of the inferior vertebra (VB). The transverse processes are depicted as T. *Panel B* is a superior view of a vertebra. ZJ designates the zygapophysial joint. C depicts a facet capsule and FP the fat pads. P are the pedicles and SC the spinal canal.

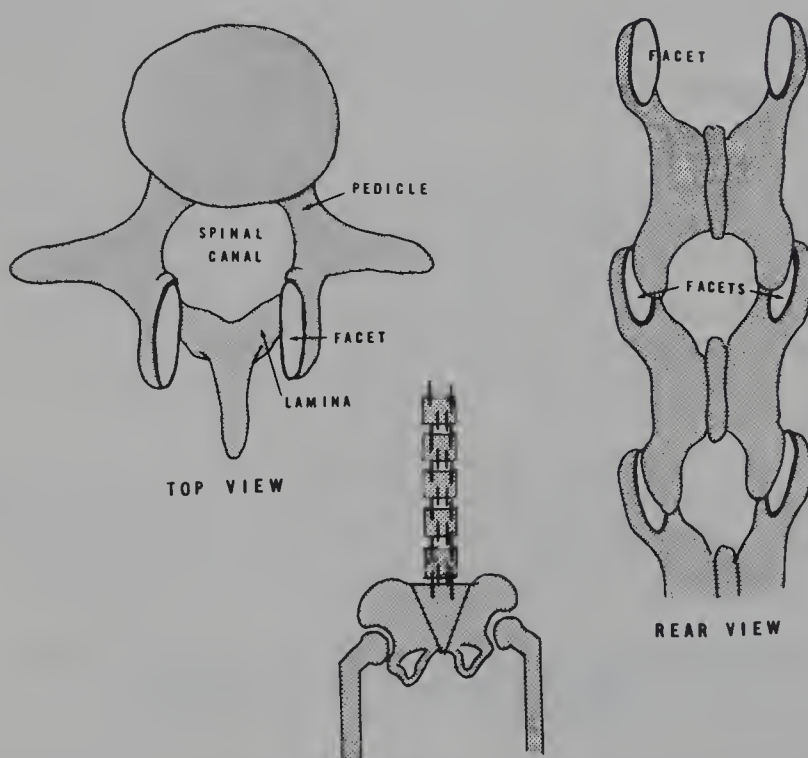


Figure 6-2. Facet alignment. The zygapophysial facets have a vertical alignment as depicted in this figure. This alignment allows flexion-extension and minimizes lateral flexion and rotation.



The facets direct the motion of the functional units, with some variation at each segment dependent upon the structure and geometric orientation at each segment.<sup>12</sup> Their vertical orientation and shape guide or restrain the movement of each segment. Their predominantly sagittal plane orientation (Fig. 6-2) favors flexion and extension but limits lateral and rotatory motion (Fig. 6-3), which protects the disk annular fibers from excessive elongation and possible tearing.

The facets are usually described as plane or flat and in a sagittal plane but this is true only of the cervical and thoracic facets. The lumbar facets are curved, albeit lying in a transverse plane. By its curve and structure the larger posterior portion of the facet lies in the sagittal plane and the anterior, or more curved portion, lies in the coronal plane.

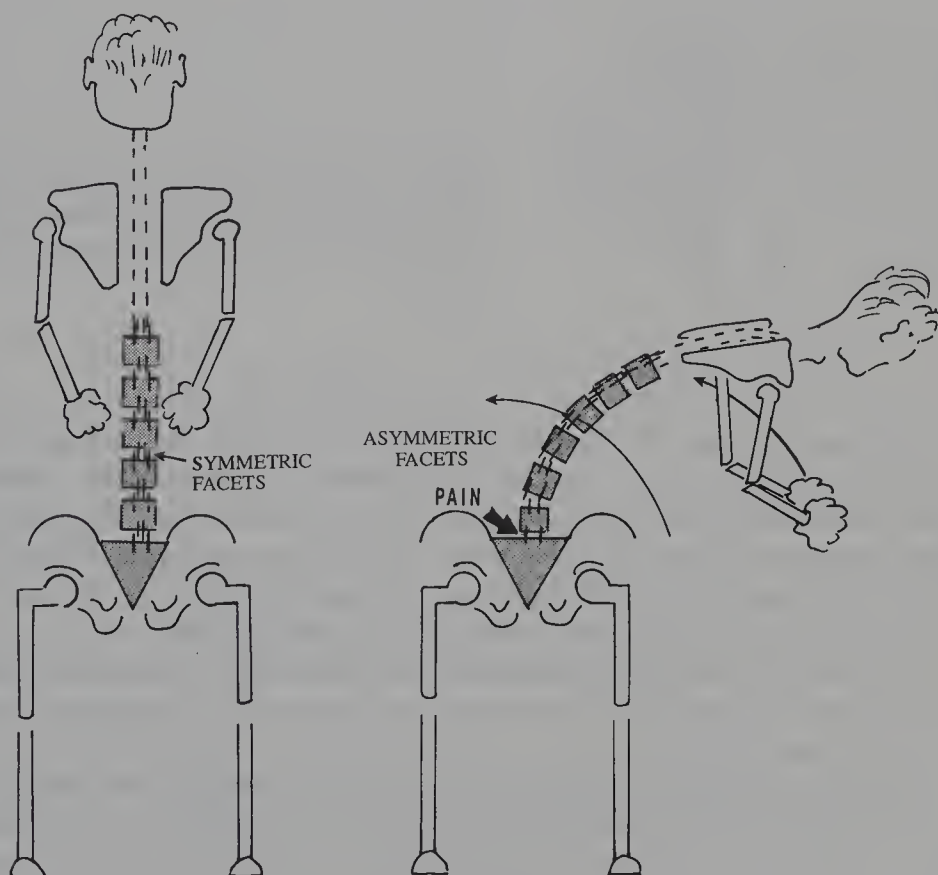


Figure 6-3. Facet guidance in flexion and flexion rotation. As viewed in the left erect posture, the facets are aligned in a vertical (sagittal) plane (*left*). When the person bends forward and to the right, the facets flex and rotate to the left (*right*). As the person reextends to the erect posture, the spine must follow the reverse paths of motion with the facets gradually derotating.

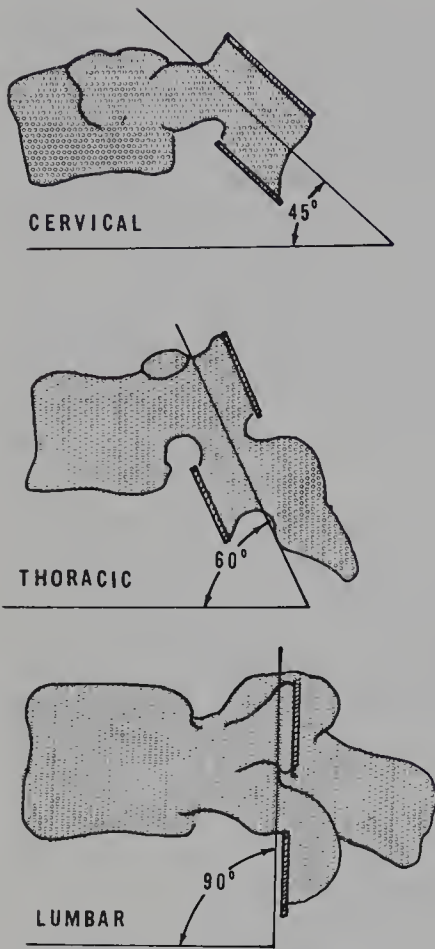


Figure 6-4. Angulation of vertebral facet planes. The angulation of the facets at the cervical, thoracic, and lumbar spine varies as does the direction of movement of each segment.

The curved facets are flat and parallel to the posterior surface of the vertebral body in their long axis. The convex surfaces of the inferior facet face forward and medially and fit congruently into the concave facets of the superior articular facet, which faces backward and medially. The anterior third of the facet lies approximately in the coronal plane and the posterior two thirds in the sagittal plane.\*

The facets change their slope as they descend from the cervical to the lumbar, being virtually parallel to the long axis of the spine in the lumbar region (Fig. 6-4).

By their angulation, they not only limit rotation but also limit translatory motion (shear). Without strong facet joints, the lumbar func-

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\*Coronal plane divides the body into front and back portions, whereas *sagittal* is a vertical plane through the longitudinal axis of the trunk, dividing the body into left and right halves.

tional units would be unstable,<sup>13-15</sup> which is what occurs from a surgical facetectomy<sup>16</sup> and in spondylolysis.<sup>17</sup>

The facets have an articular cartilage lining their surfaces that is approximately 1 mm thick. The facets have an articular capsule that is fibrous and inelastic enough to give the joint stability but lax enough to permit its physiological motion. The anterior portion of the capsule is thick and reinforced by the ligamentum flavum. The posterior portion is thin. The elasticity of the capsule is so constructed as to undergo tension during shear, which the capsule resists.

The joint capsular recesses contain fat pads, with the fat pad of the inferior recess being mostly extracapsular and communicating with the capsule through a hole in the capsule. Each fat pad is lined with synovium and is innervated. The function of the pad appears to be to dampen joint movement at their end points and supply lubrication to the joint. By being innervated, they can become inflamed and painful.<sup>14,18,19</sup>

The fat pads are restricted mostly to the upper and lower folds of the capsule and normally do not extend into the middle third of the joint. These fat pads appear enlarged in some people in what appears to be an intent to fill the gap in an otherwise redundant capsule. In older people these pads become fibrotic, especially where they appear to have been repeatedly compressed between the joint margins.<sup>20</sup> The inferior recess pad dampens extension of the functional unit as the inferior tip of the inferior articular process impinges upon the lamina below.

Lumbar motion occurs around a midline axis in the posterior portion of the disk.<sup>21</sup> In flexion and extension the inferior articular processes slide upward and downward, respectively, upon the superior articular processes of the inferior vertebra of the unit. Normally, as a person flexes, the facets disengage (Fig. 6-5). A slight degree of translation occurs, up to 3 mm being considered normal. Further flexion is prevented by increased tension in the capsule and the posterior ligaments. The compression within the articulation that restricts flexion has been measured.<sup>20,22,23</sup> The thoracolumbar fascia also exerts some restraint on flexion. In full flexion the inferior articular processes pass down like hooks behind the superior articular process and prevent further flexion and shear of the unit.

With any degree of lateral flexion in forward flexion, rotation occurs (Fig. 6-6). This rotation exerts torque on the functional unit (Figs. 6-7 and 6-8). Proper lumbar pelvic rhythm (see Chapter 1) is thus required to minimize the stress forces expended upon the facet joints, which are principally involved in these motions.

In the erect static posture there is physiological shear force expended on the adjacent facets as they approximate (Fig. 6-9).

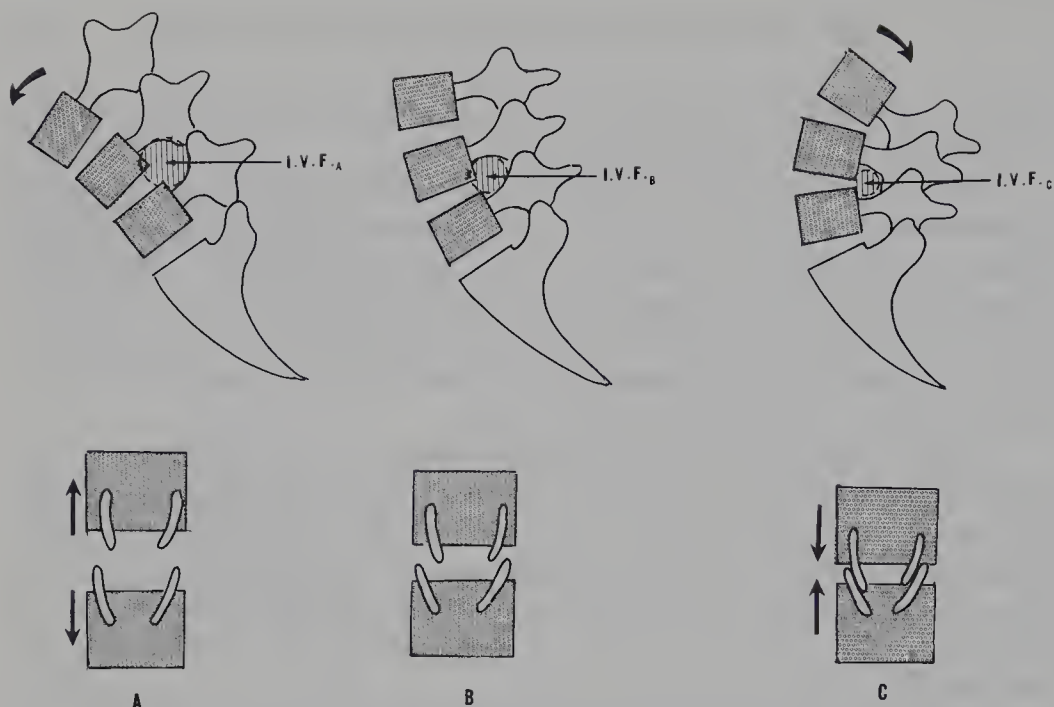


Figure 6-5. Facet movements in flexion-extension. *A*, In the flexed position the facets separate (*arrows*) and the foramina open ( $IVF_a$ ). *B*, In the static erect posture the facets are in a neutral position and the foramina are open ( $IVF_b$ ). *C*, In the hyperextended spine the facets have approximated (*arrows*) and the foramina narrowed ( $IVF_c$ ).

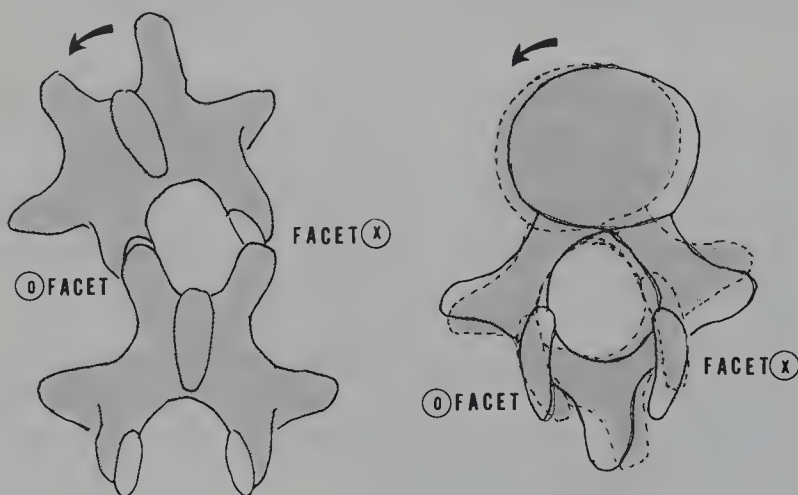


Figure 6-6. Lateral flexion rotational torque. When a person laterally flexes (bends to the side) the facets on the concave side (O) approximate and can become the axis of rotation if there is simultaneous flexion or extension. The facets on the convex side (X) separate. Rotation from this newly acquired axis of rotation causes some lateral shearing of the vertebrae (*curved arrow*). If these movements are excessive the facets on the concave side can jam, the facets on the convex side can sublux, and the disk annular fibers can be torn from shear stress.

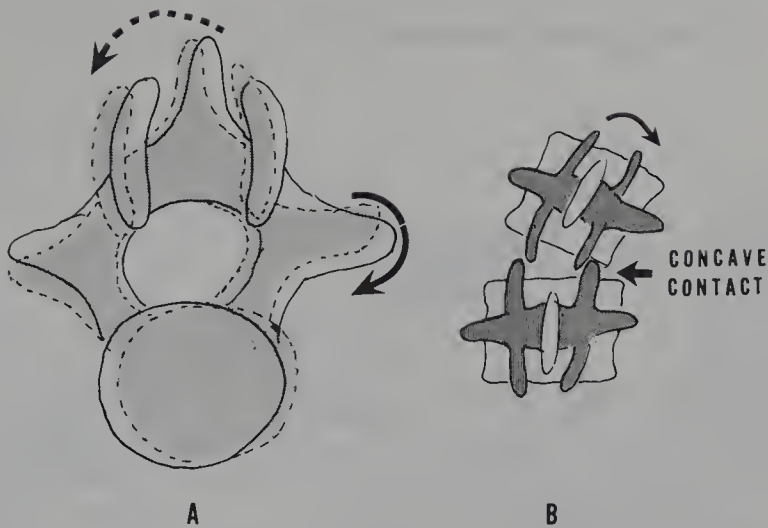


Figure 6-7. Vertebral torque in flexion-extension rotation. As depicted in Figure 6-5 the facets approximate and separate in flexion-reextension. The approximation on the concave side approximates the facets and causes a new axis of rotation (concave contact) about which the functional unit rotates (*shaded figure*). The annular fibers of the disk are laterally and rotationally extended and can tear (fail) if the torque exceeds 5 degrees.

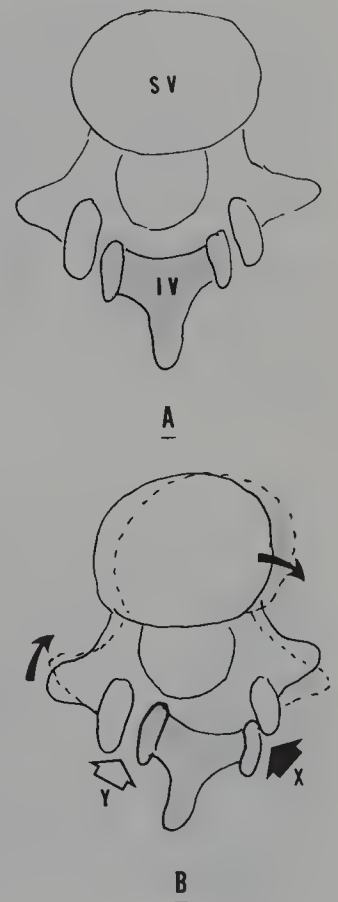


Figure 6-8. Axial rotation of a lumbar functional unit. *Panel A* depicts the normal alignment of adjacent vertebral facets. The *shaded area* depicts the inferior vertebra (IV) and SV depicts the superior vertebra. The facet alignment is shown. *Panel B* shows rotation around a new axis (X) where the facets have impinged and those on the convex side (Y) have opened. The *curved arrows* depict the motion of the superior vertebra on the inferior vertebra.



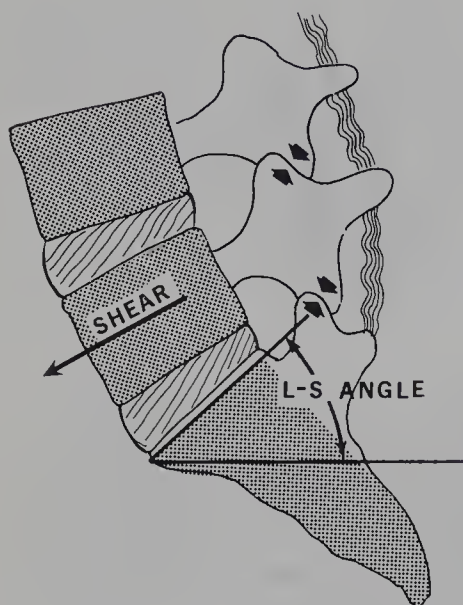


Figure 6-9. Shear forces on the lumbar functional units. The lumbosacral angle (L-S angle) of the fifth lumbar vertebra on the sacrum causes a translational (shear) movement of the superincumbent vertebrae. Excessive shear motion is prevented by the hooking of the superior facet on its immediate inferior facet (arrows).

With the facets so vitally involved in normal (physiological) movements and subjected to compressive and shear forces in inappropriate, unphysiological motions and from external stress, it is evident that, being adequately supplied with nociceptive nerves (Figs. 6-10 and 6-11), they are seriously considered as being a primary, albeit not exclusive source, of low back pain.

In the consideration of the facet involvement in low back pain, clinical evidence needs review.

Diagnostically, radiological studies have been futile, since most people, as they age, show degenerative articular changes in their facets.<sup>24,25</sup>

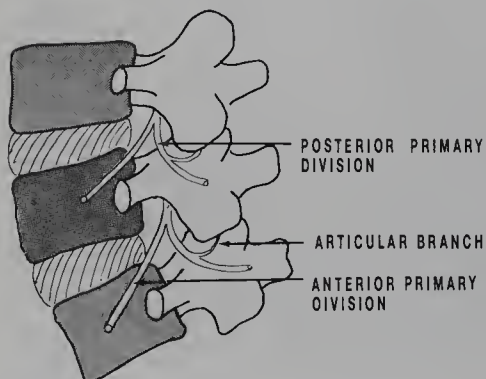


Figure 6-10. Nerve supply to the facets. A small articular branch that is sensory to the facets branches off the posterior primary division of each nerve root.

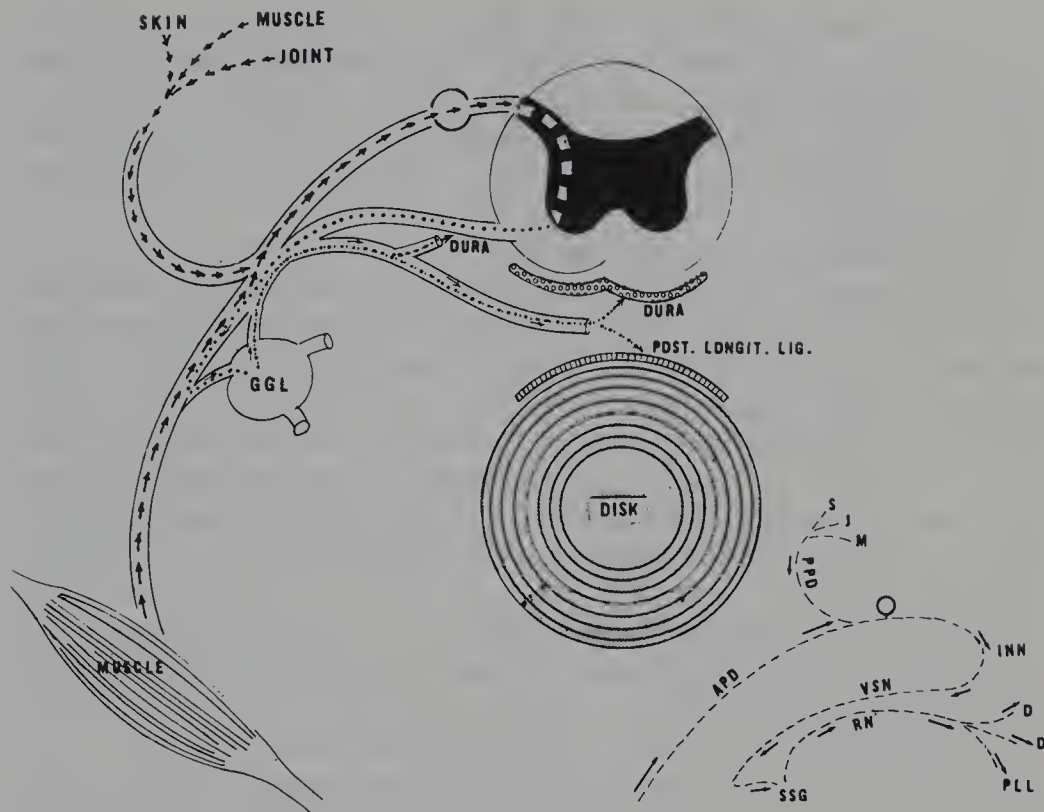


Figure 6-11. Innervation of the facet from the recurrent nerve of Luschka. The nerve of Luschka is represented here to show the articular branch of the posterior primary division (PPD) to the joint (J) in the lower dotted figure. (APD = anterior primary division; D = dura; INN = internuncial neuron; M = muscle; PLL = posterior longitudinal ligament; RN = recurrent nerve of Luschka; S = skin; SSG = sensory sympathetic ganglion; VSN = ventral sensory nerve.)

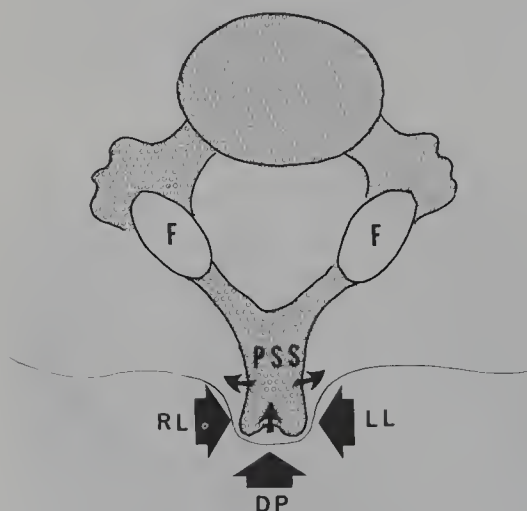
Numerous clinical tests have been postulated to designate the facets as the cause of low back pain but have not held up to careful clinical scrutiny.<sup>7</sup> A common test to confirm facet etiology of low back pain has been to bend the upright patient backward with some degree of lateral flexion.<sup>26</sup> This allegedly compresses the facets on the ipsilateral side of lateral flexion, with the patient in the extended position.<sup>27</sup> On this maneuver, the erector spinae muscles are relaxed, removing them as the cause of low back pain. Mere lateral flexion compresses the facets on the concave side but is not exclusive in asserting the facets as the cause of the resultant pain since an annular tear is often aggravated by that maneuver.

Rotation and simultaneous extension impose pressure upon the ipsilateral facets<sup>27</sup> and can be performed in the seated position as well as the standing posture.<sup>2,9</sup> Patients who benefit from a rotational manipulation that allegedly disengages the impacted facets may receive clinical benefit, but what has mechanically been achieved by the maneuver remains unclear.<sup>7</sup>

Tenderness over the inflamed facet is also difficult to ascertain as being from the facet or of the overlying erector spinae muscles. Manipulation of a single vertebra that is considered to mobilize the facets has also been advocated<sup>28</sup> (Fig. 6-12). In this manipulative technique the posterior superior spine of a specific vertebra is held between the examiner's thumb and index finger and is moved laterally and with downward pressure upon the posterior superior spinous process. Again, exactly what is moved in this procedure is unclear, and it is the patient's reaction (subjective) that determines whether the test is considered positive or negative.

The inaccuracy of the above-mentioned tests endorses the advocacy of reproducing the low back pain complained of by the patient by a provocative facet injection of an irritating substance or relief of the low back pain by a localized facet anesthetic injection.<sup>29</sup>

The inability to reproduce low back pain by precise clinical maneuvers, tests, or even by history has made the diagnosis of facet syndrome suspect and invalidated the benefit gained by facet injections or denervation. Extracapsular extravasation of the injected anesthetic agent into adjacent tissues or failure to designate the precise nerve supply to the



**Figure 6-12.** Manual maneuver of an individual vertebra. A manual diagnostic technique to determine the specific vertebra causing symptoms and to determine physiologic motion is depicted. The posterior superior spine (PSS) of each lumbar vertebra is discernable. Direct manual pressure (DP) on the process can elicit pain. Lateral motion of the specific vertebra is possible by the examiner, holding the superior spine between the thumb and index fingers and laterally moving each spine to the left (LL) or the

right (RL). The *small* arrows depict the motion elicited of the specific vertebra. This allegedly moves the facets (F) of the vertebra and localizes the pain.

facets has further confused the situation. Uncontrolled studies that eliminate placebo effect have contributed to the confusion.

The indications for facet injection have remained unclear. Selby and Paris postulated the following criteria: (1) lack of response to conservative treatment, (2) lack of neurological signs, (3) presence of low back pain with radiation to the hip, knee, and possibly the calf.<sup>30</sup> Evaluation of these criteria reveal an obvious lack of objectivity. Lack of response to conservative treatment does not implicate any precise low back mechanism, nor does it specify what the "conservative" treatment included. Lack of neurological signs again is a negative criterion and radiation into the hip, knee, and calf is nondermatomal. These vague nonspecific criteria have pervaded the literature regarding facet injections and their outcomes assessment.

Assuming that the facets are a site of nociception, denervation of these joints appeared desirable.<sup>29-31</sup> Numerous attempts were recorded and failed to substantiate this benefit.<sup>32-34</sup> Capsulotomy or denervation of the implicated facet was considered justified but was nonproductive of relief.<sup>35-37</sup> Marks et al. concluded their study with the statement, "Facet joint injection and nerve block may be of equal value as diagnostic tests but neither is a satisfactory treatment for chronic back pain."<sup>31</sup> A subsequent report of 109 patients who received (1) injections of a local anesthetic and cortisone into two adjacent facet joints, (2) injections around two adjacent facet joints, and (3) injections of physiological saline into two adjacent joints concluded "facet joint injection is a nonspecific method of treatment and good results depend on a tendency to spontaneous regression and to the psychosocial aspects of back pain."<sup>38</sup>

Another factor that must be kept in mind is that innervation of a facet joint is not from a single specific nerve root, but each facet has innervation from two levels.<sup>29,39-41</sup> One branch arises from the primary ramus of the root to that facet with another branch from the level above. Denervation must obviously approach both nerves to the facet considered involved.

The reason for giving injections into and around facet joints was the finding that pain could be elicited by injections of irritating substances into these structures with relief from intra-articular anesthetic injection. It has been known that the capsule of a facet joint accepts only 2 to 3 ml of fluid<sup>42</sup> and an injection of a greater amount possibly leaks out into the periarticular tissues.

Other techniques of treatment of recalcitrant cases of benign low back pain have been epidural injections of anesthetic agent and steroid. Epidural anesthetic injections may benefit the patient as it also anesthetizes the articular nerve going to the facets. This therefore becomes a nonspecific modality albeit beneficial.<sup>43</sup>



In a differential diagnostic testing to ascertain whether the pain emanates from the facet or the disk, a discogram can be differential. Discography is purely a diagnostic test by which the patient's pain can be reproduced from intradiskal injection, and a discogram reveals abnormalities such as degeneration and "herniation." This test is designated to reveal the cause of pain to originate from the disk when all other tests are equivocal.<sup>44,45</sup> Discography reproducing pain has revealed the presence of annular tears, which can be revealed by magnetic resonance imaging (MRI),<sup>46</sup> but reports of low back pain patients with normal MRIs have included abnormal discograms.<sup>47</sup> This test does not validate or negate the facets as the primary site of low back pain.

As the intervertebral disk is known to be innervated by the recurrent meningeal nerve (Luschka), which also innervates the facets, their relationship has been considered. This nerve supply is also approached therapeutically with epidural anesthesia and steroids. This therapy also intervenes with the nerve supply of the facets. This also allegedly could be the factor in benefiting low back pain by denervation.

Epidural injections also remain controversial, but review of summaries of the literature shows that these injections offer significant but temporary pain relief in some patients.

With the equivocal and almost negative basis for assuming that facet pathology is a major or at least significant cause of acute low back pain, do the facets play any role in this entity? The question has been appropriately raised, but the exact answer remains to be proved and universally accepted.

## DEGENERATIVE FACET DISEASE

*Degenerative arthritis, osteoarthritis, or osteoarthrosis* are terms commonly applied to a common disorder of synovial joints. It is a condition of aging that still eludes a full understanding of its etiology and development. The common label of *benign arthropathy of inevitable aging* has been of little comfort to the affected patient.

The presence of degenerative changes of the facet joints without pain is a frequent finding that has postulated that this is a natural sequelae of disk disease from anterior narrowing of the functional unit.<sup>47</sup>

The structure, chemistry, and function of normal cartilage must be understood before pathological changes and their sequelae can be appreciated.

Cartilage nutrition is maintained by infusion and diffusion of fluids (Fig. 6-13). The structure of normal cartilage makes this nutrition possible in that normal cartilage is composed of collagen fibers (Fig. 6-14) within a mucopolysaccharide matrix.



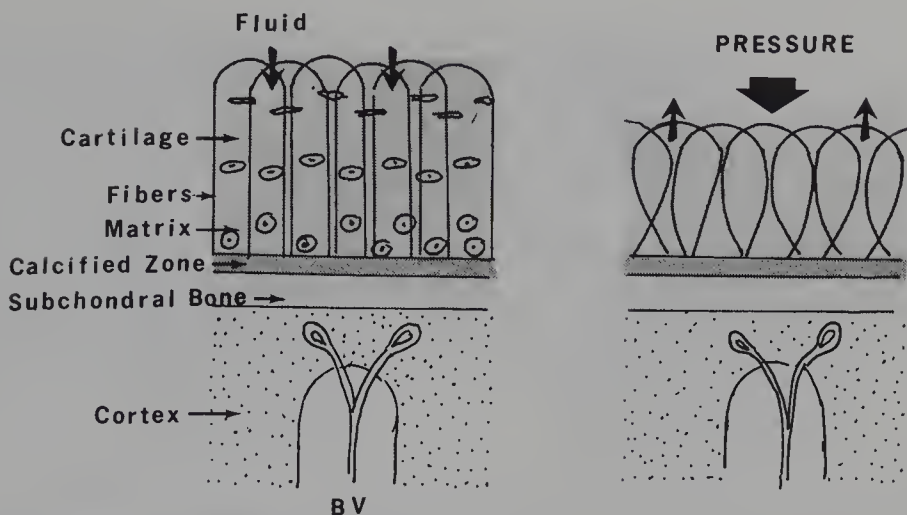


Figure 6-13. Cartilage nutrition. The “relaxed” cartilage has collagen fibers and chondrocyte cells within the matrix (*left*). These cells are active in the basal layers and flatten towards the periphery until they flake off and exit the cartilage. Immediately below the cartilage is the calcified zone that does not permit the passage of blood vessels (BV) into the cartilage. Immediately under this zone is subchondral bone and then the cortex. The blood vessels end as bulbs. In the relaxed state the cartilage is expanded and absorbs fluid (*small arrows*) from the synovial cavity. The cartilage is compressed by gravity or muscular contraction (*right; large arrow*). Compression occurs due to the flattening of the cartilage fiber springs. The fluid compressed from the cartilage weeps into the synovial cavity (*small arrows*).

The collagen fibers are coiled, allowing them to act as compression springs that compress from external pressure (Fig. 6-15), with a resultant narrowing of the cartilage. Removal or decrease of the external pressure allows these collagen springs to reextend and allows the cartilage to regain its normal width. This compression and relaxation is the physical basis of imbibition.

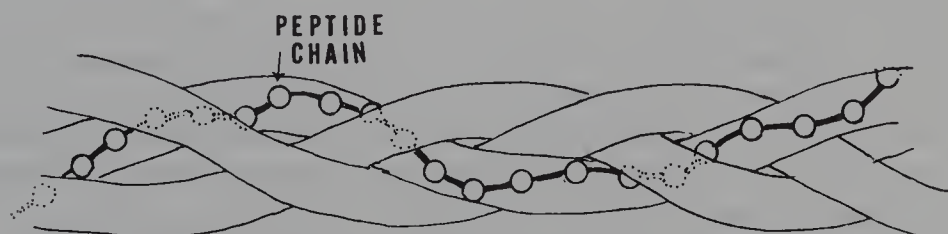


Figure 6-14. Tropocollagen trihelix fiber (schematic). This type I collagen molecule is a trihelix peptide chain composed of two alpha-1 and one alpha-2 peptides in which every third molecule is a glycine amino acid. The three intertwining peptide chains form a trihelix collagen fiber. (Modified from Alberts, B, et al: *Molecular Biology of the Cell*. Garland. New York, 1983, p 694.)

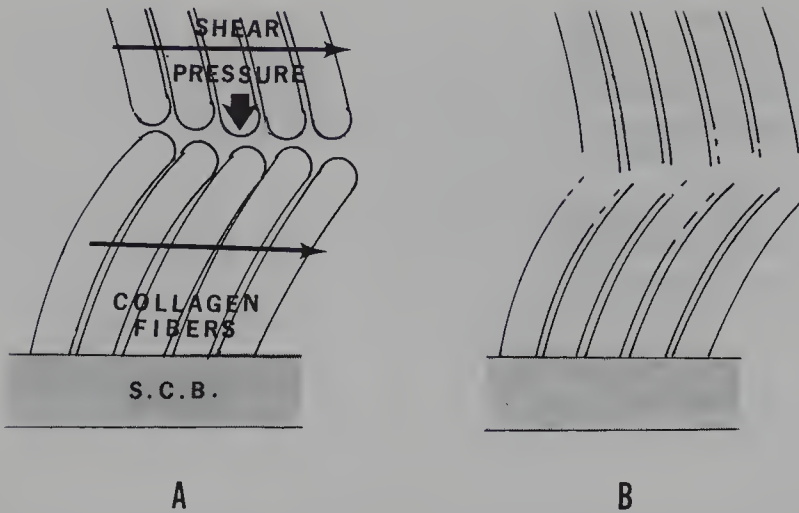


Figure 6-15. Shear deformation of cartilage. The opposing cartilages of a moving joint cause the curving deformation of the collagen fibers (A). This shear force is augmented by compressive forces from gravity and muscular action. The underlying subchondral bone (SCB) is shown. The shear effect has caused a degeneration of the collagen fiber with irregularity of the cartilage matrix (B).

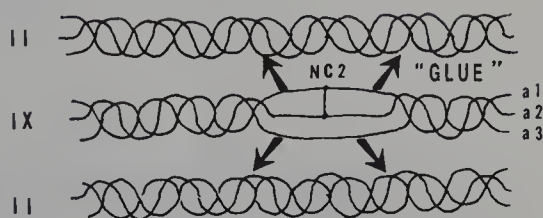
Articular cartilage consists of cellular and extracellular components, with the extracellular components being collagen, proteoglycans, and other proteins. The cellular components are chondrocytes (5% of the total volume). The proteoglycan matrix is negatively charged and is hydrophilic (imbibes water), which encourages diffusion of nutrients. Along with the enclosed collagen fibers, it affords tensile strength to the cartilage.

The collagen fibers are of type II, IX, X, and XI of the 14 types currently recognized (Fig. 6-16).

The collagen fiber molecule is composed of three polypeptide chains, with every third residue being amino acid glycine, and the others, the X and Y fibers, being proline and hydroxyproline. The chains are termed *alpha*, with each chain being genetically determined. Each trihelix chain is connected to its adjacent chain by a water molecule (amino acid hydroxy group).<sup>48</sup> These intermolecular cross-links are also the sites of glycosylation, which is involved in the nutrition and remodeling of the collagen fibers.

The structure of type IX collagen fiber differs fundamentally from type II fibers and is considered to be the “glue” that holds all the type II fibers together.<sup>49</sup> Degradation of these fibers is considered to be the basis for degenerative arthritis.

Figure 6-16. The role of type IX collagen in cartilage. There are predominantly type II, IX, X, and XI collagen fibers in cartilage. Genetically determined type IX has been hypothesized as being the glue that holds the type II fibers together. Degradation leading to degenerative changes has been thought to represent ungluing of the collagen fibers, weakening the structure of the cartilage. The cause of this ungluing remains obscure.



Degradation of articular cartilage occurs from release of proteolytic enzymes from chondrocytes, synovial cells, and neutrophils.<sup>50</sup> These proteinases are termed collagenase (destroys collagen), stromelysin (destroys matrix), and neutrophil elastase (destroys elastin).<sup>51</sup> These enzymes are allegedly regulated by an inhibitor metalloproteinase (TIMP). When there is an imbalance of TIMP to the proteinase, degenerative changes occur in the cartilage.

The lubricant expressed from cartilage upon pressure is hyaluronidase, which minimizes friction but also acts as an adhesive, keeping the articular surfaces together. Compression of the cartilage flattens the coiled collagen fibers, whereas shear, which is a frequent occurrence of articular joints, deforms the angulation of the collagen fibers (Fig. 6-17). Contrary to widely held assumption, friction's effect on cartilage has been declared as negligible.<sup>52,53</sup>

Degenerative cartilaginous changes are thus considered to occur from:

1. Longitudinal forces, which include direct mechanical impact and muscular contraction
2. Unphysiological and excessive compressive forces on the cartilage
3. Impact on the subchondral bone, with resultant microfractures

These traumata alter the metabolism of the cartilage,<sup>54</sup> changing the pore size of the matrix components and causing an outflow of matrix, which alters the osmotic pressure of the cartilage. The matrix is also altered by the action of lysosomal lytic enzymes.

Studies on trauma to cartilage have focused on the repair mechanisms following lacerations (Fig. 6-18) in laboratory animals. Superficial lacerations that do not approach the subchondral bone do not progress or heal. Deep lacerations that violate the underlying bone plate undergo characteristic changes.

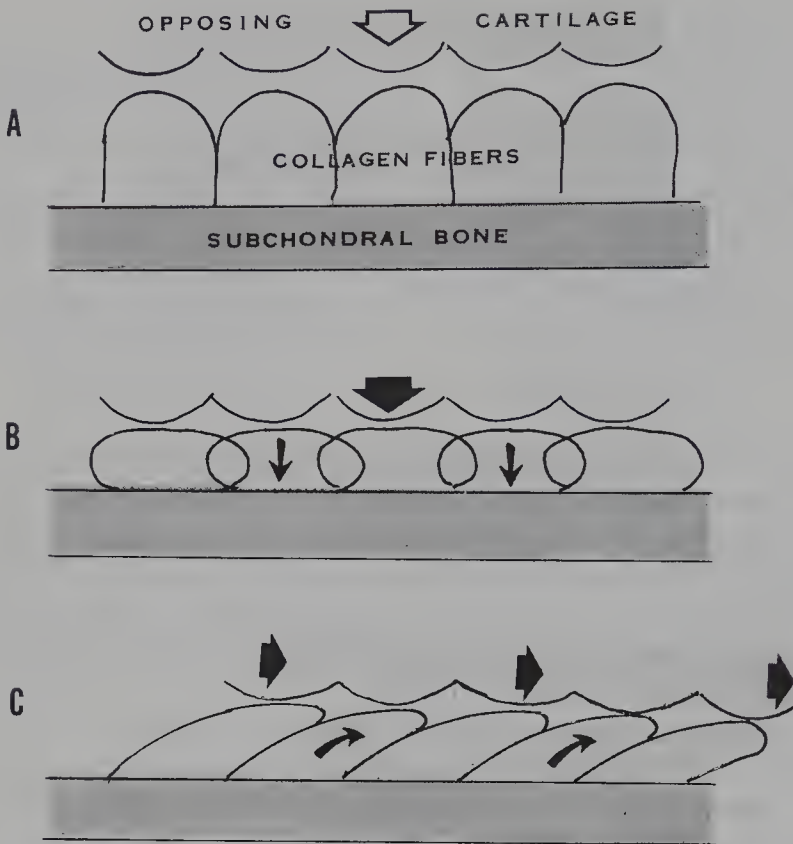


Figure 6-17. Shear deformation of cartilage. Opposing cartilages of a moving joint cause a curving deformation the collagen fibers (A). This shearing force is augmented by compressive forces from gravity and muscular action (B). The underlying subchondral bone is shown. The shear effect has caused a degeneration of the collagen fiber with resultant irregularity of the cartilage matrix (C).

Initially the defect fills with blood cells from the endplate blood vessels, which immediately becomes organized into a fibrous clot. Within 10 days, some of the blood cells change into fibroblasts. Chondrification of the tissue forms fibrocartilage, which fills the defect, forming a “dimple” within the surrounding normal cartilage.

There is a concomitant increase in the proteolytic enzymes within the cartilage, causing a decrease and chemical change in the proteoglycans.<sup>55</sup> There is an increase in water content that remains unexplained.

Initially, in degenerating cartilage there occurs a flaking of the superficial surface. Cysts form in the tangential layers, which open into the joint surfaces, causing craters. Hyaluronidase and other enzymes penetrate into this crater, causing loss of chondroitin, which is a material component of cartilage matrix.



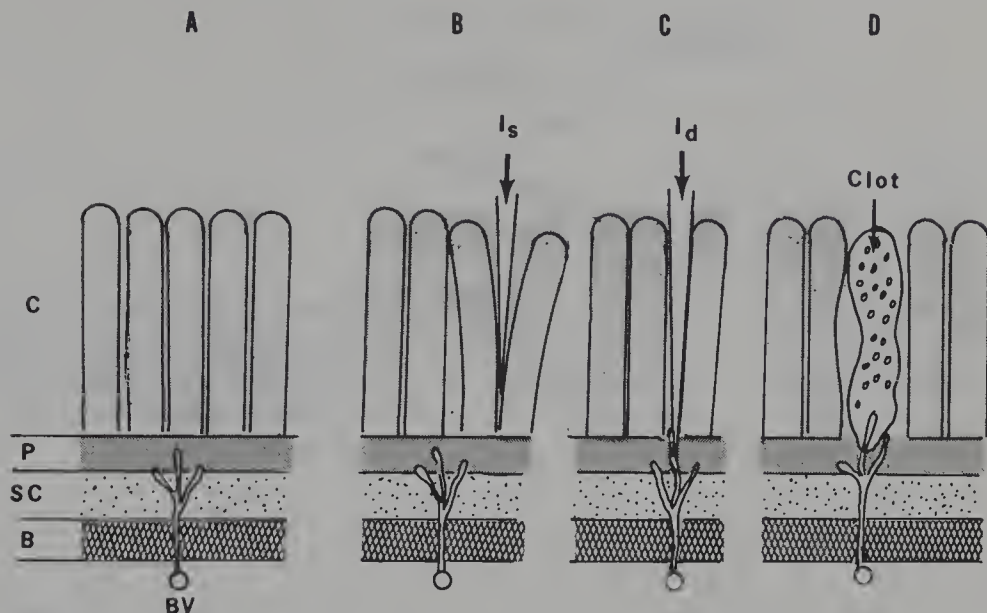


Figure 6-18. Response of cartilage to laceration. *Panel A* depicts normal cartilage (C) with a calcified plate (P). Below the plate is the subchondral plate (SC) and bone cortex (B), containing blood vessels (BV). *Panel B* depicts a shallow laceration ( $I_s$ ) that does not reach the subchondral bone and does not heal. *Panel C* depicts a deep laceration ( $I_d$ ) that penetrates the matrix and enters the subchondral bone plate, causing invasion by blood vessels. *Panel D* depicts the penetrating blood vessels that form a clot, containing fibroblasts, which cause healing by fibrocartilagenous tissue.

A loss of elasticity results, and the synovial lubricants lose their viscosity, adding further mechanical trauma to the joint. As the loss of cartilage occurs, the subchondral bone fills the denuded areas until gradually the opposing articular surfaces are separated by bone, fibrocartilage, and denuded cartilage.

Degenerative changes within the functional unit undergo a sequence of degeneration termed *spondylolysis* (Figs. 6-19 and 6-20). With the attention on disks, there also occur degenerative arthritic changes in the facets. In the sequelae, narrowing of the foramen (Fig. 6-21) can cause nerve root entrapment, with neurological impairment. When the contents of the foramen (Fig. 6-22) are encroached upon, it leads to neurological deficit.

Unilateral facet hypertrophy from degenerative changes can impinge on the foramen, with entrapment of the nerve root (Fig. 6-23), especially when combined with thickening of the laminae.



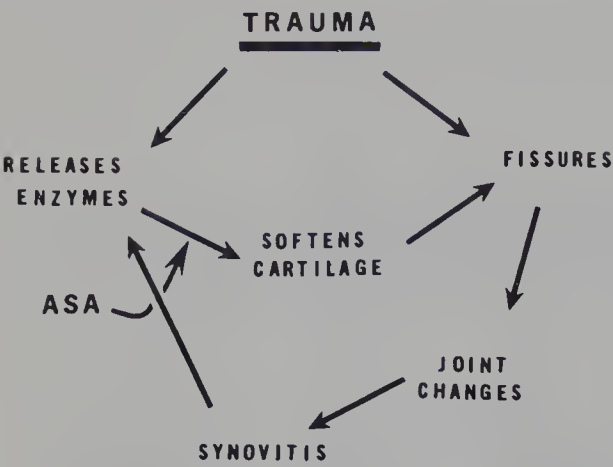


Figure 6–19. Schematic process of degenerative arthritis. Schematic changes that lead to degenerative arthritic changes.

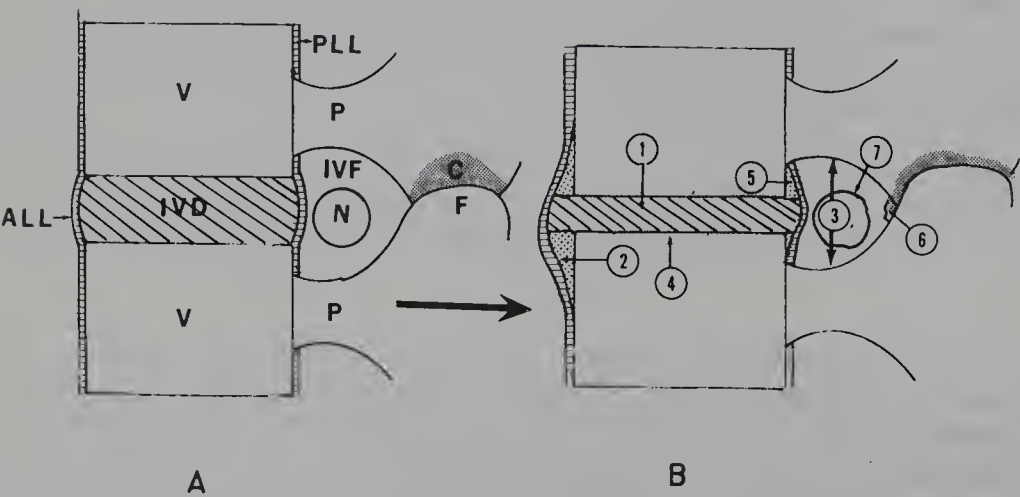


Figure 6–20. Sequence of degeneration: spondylosis. *Panel A* depicts a normal functional unit. (V = vertebra; ALL = anterior longitudinal ligament; IVD = intervertebral disk; PLL = posterior longitudinal ligament; IVP = intervertebral foramen; P = pedicle; N = nerve root; and C = cartilage of the facet [F].) *Panel B* depicts stages of degeneration: (1) narrowing of the disk, (2) formation of an osteophyte, (3) narrowing of the foramen, (4) sclerotic endplate changes, (5) separation of the posterior longitudinal ligament from the vertebra, (6) degenerative changes of the cartilage of the facet causing (7) foraminal stenosis leading to nerve root compression.

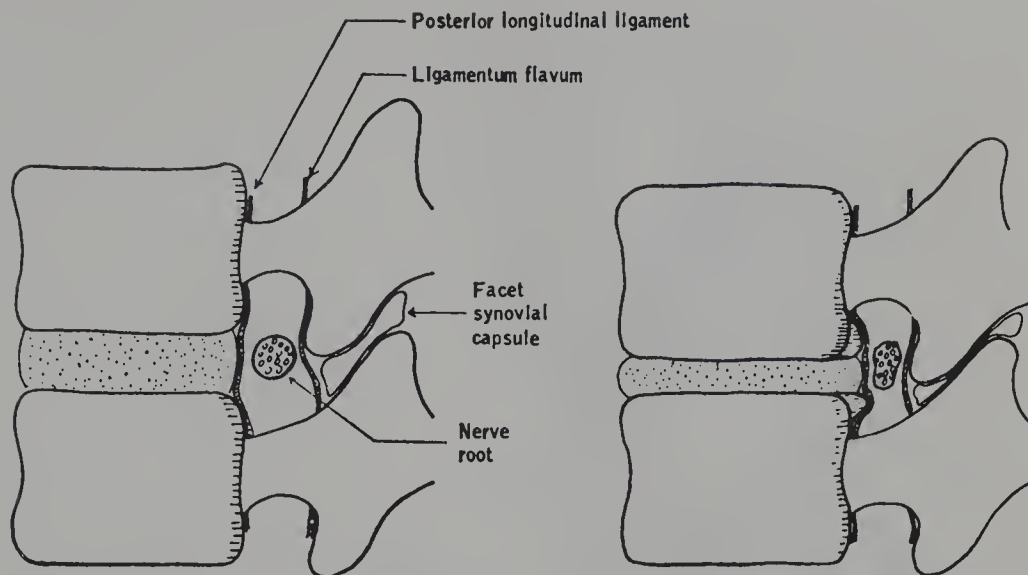


Figure 6-21. Intervertebral foramenal nerve root impingement. The *left* drawing depicts a normal side view of the foramen and enclosed nerve root. The *right* drawing depicts the degenerative changes, including osteophytes, thickened posterior longitudinal ligament, and facet changes that narrow the foramen.

Disk degeneration, causing spurring of the vertebral osteophytes, increases foraminal stenosis from facet arthritis (Figs. 6-24 and 6-25).

Foraminal stenosis can occur from tropism of the lumbosacral spine (see Fig. 5-20). The condition termed *tropism* is an asymmetrical segmental scoliosis usually found in the lower lumbar segments L4-5 or L5-S1. Normal spinal functions are impaired, as the facets are off center and weight bearing is not equally borne upon the disk anteriorly or the facets posteriorly. The three-legged stool concept of the functional unit is impaired.

On spinal motion the axis of rotation of that segment resides on the concave side, where overriding facets can become "locked." The nerve root on that concave side is prone to entrapment. Diagnosis of this condition is by radiological studies when the condition is clinically suspected. It may be incidentally found in an asymptomatic person.

Severe facet regeneration can result in a bridging between adjacent facets and immobilize that spinal segment (Fig. 6-26).

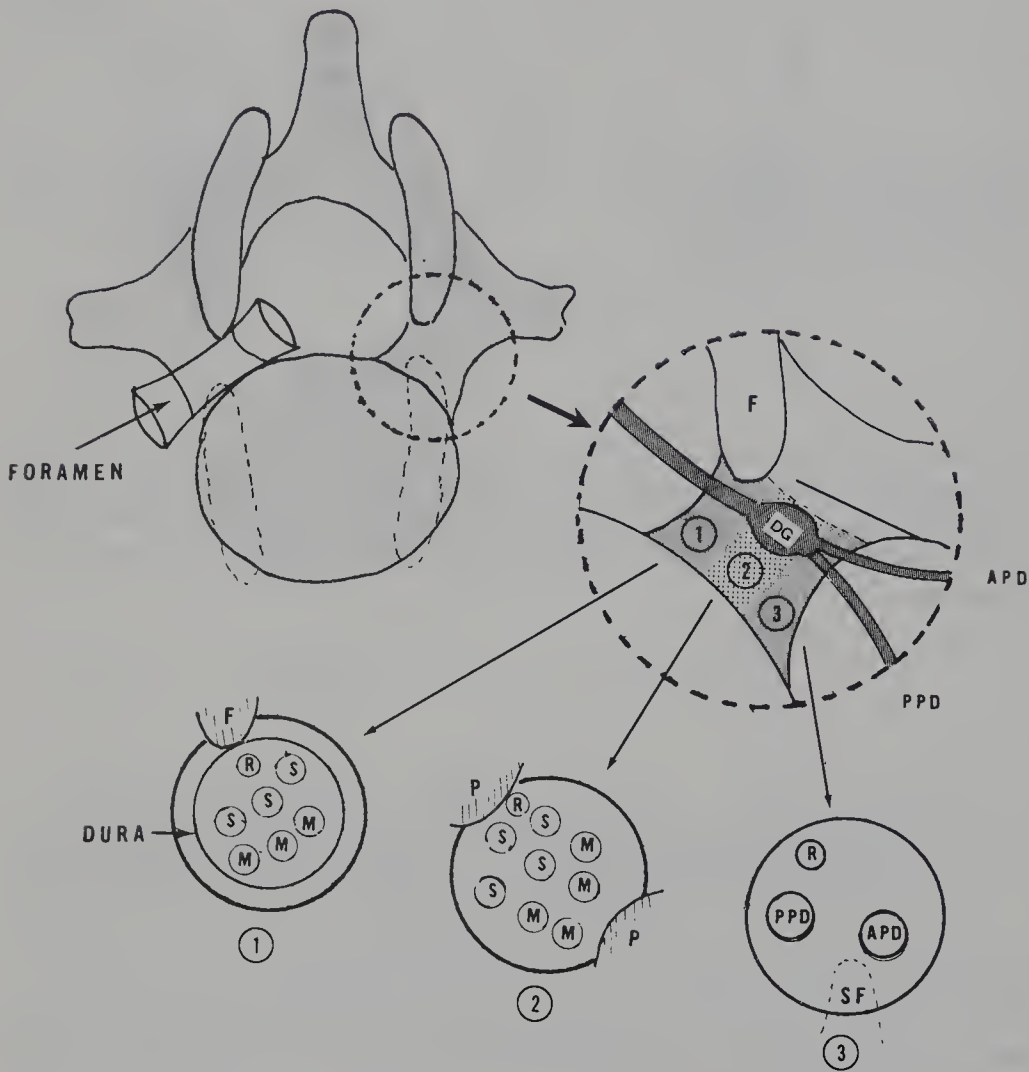


Figure 6-22. Contents of intervertebral foramina. The foramen is pictured as a funnel-shaped opening. The length of the canal is 3 to 5 centimeters. In the large dotted circle the canal is divided into three portions: the inner (1), middle (2), and the outer (3). The dorsal root (DG) is shown in the second area here with the facet (F) in the (1) area. The contents of each area (1, 2, and 3) are shown below with the nerve root contents. (S = sensory; M = motor; R = recurrent Luschka nerve; PPD = posterior primary division; APD = anterior primary division, and SF = spinal fluid.)

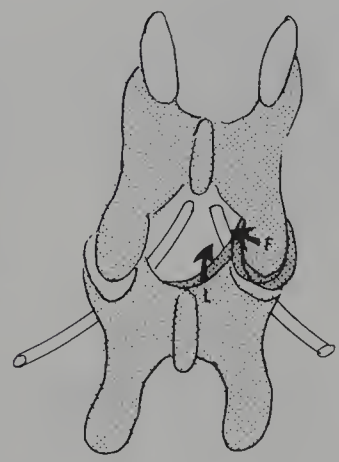
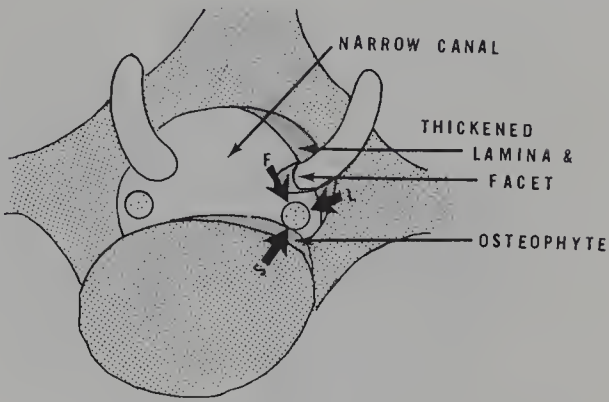


Figure 6-23. Unilateral degenerative facet disease with nerve root entrapment. Upper view shows hypertrophy of the facet (F) and the lamina (L) pressing on the nerve root. Anteriorly there is the formation of a spur (S) otherwise termed *osteophyte*.

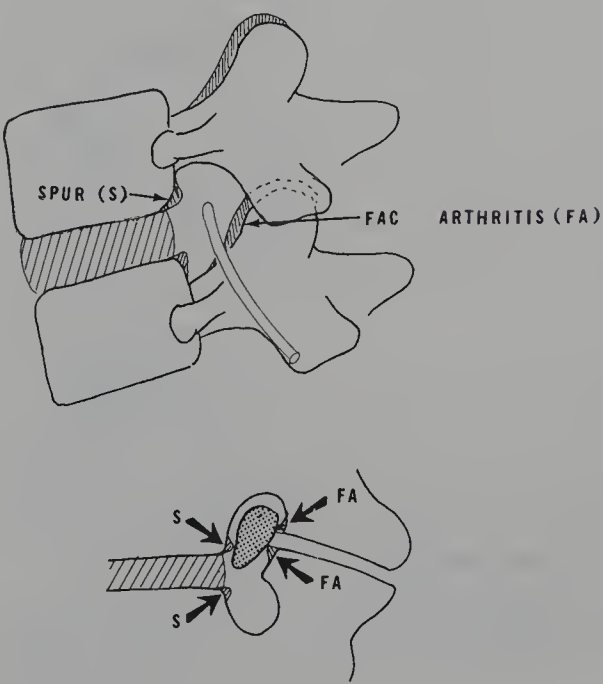


Figure 6-24. Foramenal stenosis from disk degeneration and facet arthritis. The *upper view* is a side view of the foramen with osteoarthritic spurs (S) and degenerative changes of the facet (FA). The *lower view* is an enlarged view of the foramenal stenosis from spurs (S) and facet arthritic changes (FA).

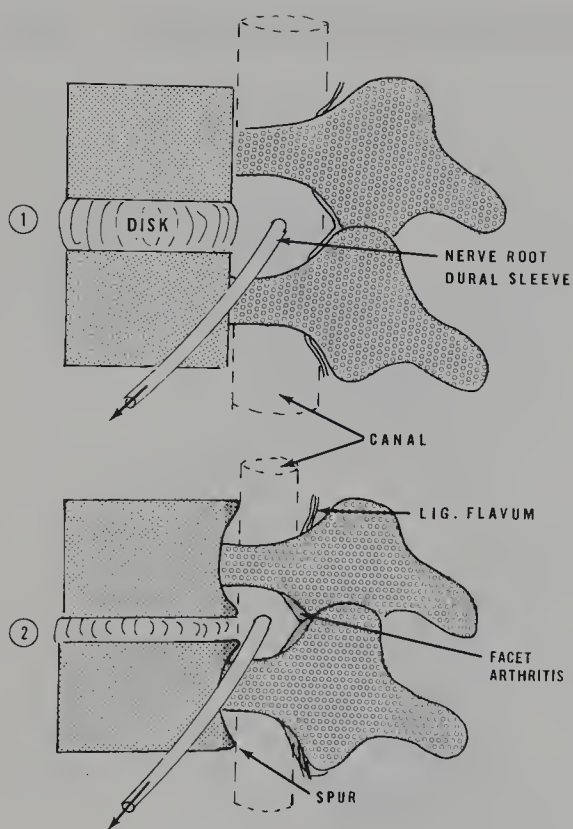


Figure 6-25. Foramen and canal stenosis: side view. The *upper view* is a normal functional unit. The *lower view* shows a narrowed canal and foramen from degenerative changes.

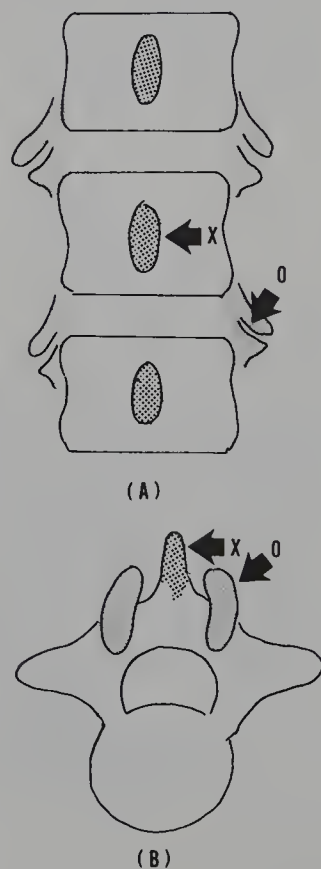


Figure 6-26. Fusion of facet joints. *Panel A* shows that diagnosis of a fused facet (O) is by manual lateral manipulation of the posterior superior spine (X). *Panel B* depicts the superior view of the same procedure.



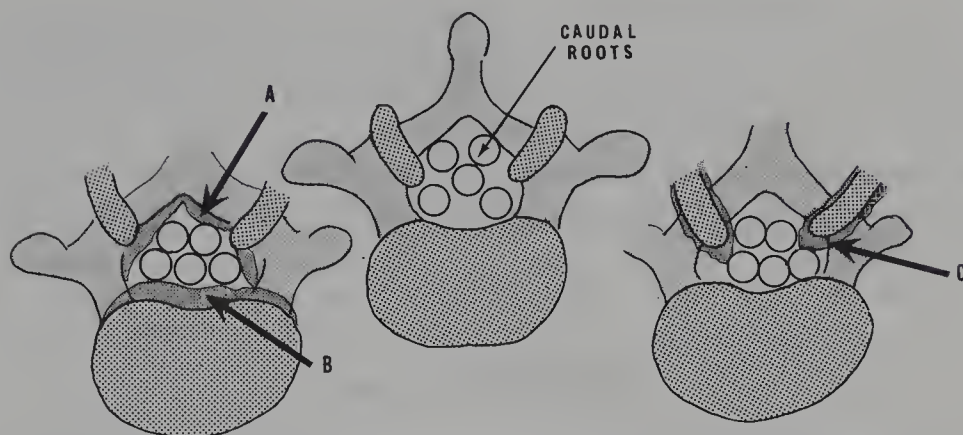


Figure 6-27. Spinal stenosis. The central figure depicts the caudal roots within a normal spinal canal. The left figure depicts total encroachment of the roots from hypertrophy of the lamina (A) and the posterior aspect of the vertebra (B). The right figure shows encroachment of the root from hypertrophy of the facets (C).

Significant bilateral disk degeneration with osteophytosis and bilateral facet hypertrophy combined with laminal hypertrophy (see Fig. 5-16) can lead to spinal stenosis (narrowing of the spinal canal) (Fig. 6-27). In this condition the caudal equine roots are entrapped below that level. The diagnosis of this condition is established by a history of pseudoclaudication, neurological impairment, and confirmation by computerized axial tomography (CAT), MRI, or myelography.

Degenerative disease of the facets is so prominent in the aged spine and after repeated trauma that its consideration as a major cause of low back pain is understandable. Degenerative change in the facets, however, is so frequently found without pain that the question of pertinent involvement remains questionable. Only when there are neurological symptoms is the condition seriously considered, as the treatment is surgical.<sup>37,56,57</sup>

Single leg pain that is clinically attributable to the facet pathology, with pain more severe than low back pain, usually of long duration, without necessarily an acute onset, and unrelieved by bed rest, has become diagnostic. Clinically there is also limited low back extension, with aggravation of the leg pain from this maneuver.

Treatment becomes indicated when the condition is not self-limited and progresses gradually with objective findings. Pain can often be ameliorated by an epidural steroid injection, and pain should not be the sole criterion for surgical intervention.<sup>56</sup>

Facets are richly supplied by nerve fibers,<sup>1,7,37,58-60</sup> which justifies considering the facets as sites of nociception. Somatic nerves have been documented within the facets, but the presence of autonomic nerves implicates sympathetic nerves also subserving as nociceptors.<sup>61</sup> This may indicate the benefit of sympathetic blocks relieving low back pain, even though neurotransmitters are also found in the posterior longitudinal ligament.

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## CHAPTER 7

# Quantitative Measurements

The determination of the cause of back pain from the patient's history and physical relates it to muscular, mechanical, neurogenic, or pathological causes.<sup>1</sup> Most low back pain relates to soft tissues, and injury to these tissues is difficult to document. Therefore, the treatment has been empirical. No veritable site of tissue injury can usually be ascertained in patients with low back injuries, thus the soft tissues must be impunged. The primary focus now is changing from relieving pain to restoring function.<sup>2</sup>

Whether the injury is considered to be to disk, ligament, muscle, or tendon, complex exercises have been empirically prescribed with increasing frequency for the treatment of benign low back pain.

On the basis of the current sports medicine approach on neuromusculoskeletal injuries, performance has become the basis of evaluation and intervention. Performance has, in turn, been equated to strength, endurance, neuromuscular control, and motivation.

Many evaluation systems have been propounded to quantitate strength factors and range of motion and effort. In the third edition of the *American Medical Association Guide to the Evaluation of Permanent Impairment*<sup>3</sup> range of motion for evaluating lumbar capacity with the use of an inclinometer is advocated. This, at best, is crude, inaccurate, and unreliable as a criterion of function, yet remains a measurement.

In the late 1960s control of the speed of muscular contraction was proposed as being valuable.<sup>4</sup> This was considered to be isokinetic, as the generated torque allowed the force to be measured throughout the range of motion by controlling the velocity. This evaluation proved of value in sports injuries to the extremities, but it was only applied to the low back in 1980.<sup>5</sup> This original testing was in the recumbent position.



Equipment was modified to test the low back in the sitting position.<sup>6</sup> This equipment merely confirmed that the torso strength of men was greater than that of women and that the extensors were significantly stronger than the flexors. Physical fitness and size of the person were not computed.

Isokinetic testing in standing was initiated by Mayer et al.<sup>7</sup> in persons with and without low back pain. It was in these studies that the extensor back muscles were found to be weaker in persons with low back pain, but the results confirmed that “willful effort” influenced the outcome of the test. It also tested only sagittal forces, whereas these forces were being measured in the sitting position.<sup>8</sup>

Normally, rotational torque is about 50% of extensor torque and is significantly decreased in patients with low back pain (65% of extensor force). The immobilized pelvis in most testings violated the normal mechanics of lifting assumed by most individuals. More recent equipment allows the person to lift in any posture desired. This simulated testing revealed that patients with low back pain lifted with 67% of normal values.<sup>9</sup>

More recently, an accurate evaluation system providing quantitative data has been developed “to clarify these subjective complaints and objectively verify their presence.”<sup>10</sup>

A back evaluation (B-200) system that measures “absolute values” such as isometric torque in pounds per feet and range of motion in degrees to assertedly characterize a person’s function “has not found a significant difference in isometric torque between back pain patients and normal individuals,”<sup>11</sup> although speed of performance was a major discriminator between individuals with back pain and those without pain.<sup>12</sup>

Increased fatigue has confirmed substitution of normal muscular function and a “deviation in the arc of motion” as contributing to dysfunction. Studies have ascertained differences after periods of prolonged rest.<sup>13-16</sup>

Differences in range of motion as related to dysfunction of motion with or without pain are also unclear.<sup>14,17,18</sup>

Velocity is an important measure of back function, but velocity is difficult to objectively measure.<sup>19-24</sup> All these factors—*isometric torque strength, range of motion, and velocity*—have been designated as the parameters to be objectively designated in measuring spinal function. All these factors must also be applied to task specifics of daily activities and occupational activities.

In simple unconstrained isometric testing, the posture of the individual is difficult to control or even incorporate into the evaluation. Other factors of the total person in the act of lifting, such as arm strength and leg strength as well as trunk strength, must be incorpo-

rated into each occupational requirement.<sup>25</sup> The coordination of the movement must also be ascertained.

An isometric lumbar test<sup>26</sup> that requires the subject to hold the trunk parallel to the floor while the torso is unsupported and measure the time before fatigue has been correlated with an increased incidence of back injury if below 1 minute. This test implicates muscle strength (isometric) and endurance.

Of many instruments, the B-200 type equipment was developed for some of these measurements and merits evaluation.<sup>10</sup> The B-200 (dynamometer) is a triaxial dynamometer that measures torque, velocity, and position of the trunk. It measures isoinertial movement signals at the rate of 50 Hz via an IBM-XT computer nine channels.<sup>20</sup>

There are several factors mandated in the use of this machine that merit discussion. The "patient must be safe during the testing." This implies that movements and functions must not be "unphysiological" and that the patient not be fearful of injury or reinjury.

The absolute values desired are variable rather than absolute, as their interpretation allegedly evaluates motivation, which involves pain, interpretation of pain, and psychological factors, as well as physical ability.

Muscular back pain secondary to a sprain or strain is claimed to be the most common category of low back pain,<sup>25</sup> yet there are few if any precise objective signs. Mostly there is a resultant limited function attributable to "protective spasm" and/or tenderness to palpation. The spasm is also debatably either neurological or psychological.

Definitions of muscle activity merit review:

*Isometric* is a contraction of muscle in which its length remains constant.

*Isokinetic* is a contraction of muscle in which the length shortens or lengthens.

*Isoacceleration* is a condition in which acceleration remains constant.

*Isoforce* measurement is a condition in which the muscular tension (force) remains constant. Also termed *isotonic*.

*Isoinertial* measurement is a condition in which the muscle moves at a constant mass.

The reliability of isometric torque measurements has been good in normal patients but not yet clarified in patients with low back pain.<sup>26</sup> The B-200 has poor reliability in sagittal extension<sup>27</sup> and in documenting range of motion,<sup>28</sup> as have most other techniques of measurement.

In clinical diagnostics it has been considered difficult to document measurements of a lack of effort.<sup>26</sup> Neither has it been possible to validate effort in outcomes assessment.

Combined with an electromyographic input identifying specific muscles, such an instrument has research potential. Without this addition, it measures only in the upright position and not in forward flexion and rotation, which are considered predominant in low back pathology.

If we assume that acute low back pain without structural lesion is a functional disorder, evaluating malfunction is apparently necessary.<sup>29</sup> Function has been equated as range of motion, strength of trunk muscles, and velocity in well-defined motions. Pre- and postevaluations of drugs used in treating patients would be welcome, especially to determine if specific medications benefit these three factors. In patients with decrease in force<sup>30-32,37</sup> and speed of function, objectively documented retesting after medication would objectively document this functional restoration. Studies so far indicate that velocity is a more sensitive marker of impairment.<sup>29</sup>

## RANGE OF MOTION

Measurement of spinal motion has been considered important in evaluating clinical disabling entities, but determination and quantification have eluded most clinicians and researchers.<sup>28</sup> Motion of the spine occurs about several motion segments. Around different axes of rotation, motion differs at each level and there is no simple motion, as flexion-extension occurs in conjunction with rotation.<sup>32</sup> There is also translation of each segment.

Range of motion is traditionally recorded as the end points of rotation around three axes. These three motions are flexion-extension (about a transverse axis), lateral bending (about a sagittal axis), and axial rotation (about an axis parallel to the longitudinal axis).

Axial rotation is a more questionable variable in testing, in fact, a variable motion as to causation<sup>32</sup> of pathology or functional restriction. Measurement of axial rotation remains difficult.

Range of motion measurement techniques have included goniometers,<sup>33-37</sup> skin markings,<sup>38,39</sup> flexible rulers, floor-to-finger distances,<sup>40,41</sup> and various instruments such as a spondylometer,<sup>41,42</sup> kyphometer,<sup>43,44</sup> and a sophisticated instrumentation called the *spinograph*.<sup>45</sup>

All these instruments have suffered failure of reproducibility in their use in pre- and posttesting and from various examiners on the same patients. Probably more significant is the relationship to variations in range of motion as a precise diagnostic factor. Which tissues are respon-

sible for limitations, at which spinal level and causing which symptoms, remains conjectural. The findings<sup>28</sup> that current instruments are no more accurate than any of the other techniques are therefore of little significance in diagnosing pathomechanics of low back pain.<sup>46</sup>

Fatigue has been considered a significant factor in faulty function, resulting in pain and dysfunction.<sup>47-51</sup> Fatigue in industrial workers has been a major factor in reducing their skill and predisposing them to injury.<sup>52</sup>

In motor activities there are numerous types of muscular contractions in effect, especially in trunk muscles.

Isoinertial contractions are the least studied, yet probably the most commonly experienced. In a muscle generating torque equal or less than the resistance, the muscle does not change its length, whereas if the torque exceeds the resistance, the muscle length changes, with the difference (surplus) torque determining the velocity. This force is probably effective<sup>53</sup> in the deceleration of the erector spinae muscles discussed in low back mechanics.<sup>54</sup>

Kinetics involved in any motor activity recruits  $\alpha$  and  $\gamma$ -motoneurons, spindle system, and peripheral proprioception as well as motor patterns engrammed in the cortex.<sup>55-60</sup>

To further complicate the activity of trunk flexion-extension, rotation, and derotation in bending and lifting activities, there is the participation of ligaments and fascia and specific bony structures with different angular distances of rotation.<sup>61,62</sup> Load during extension is transmitted through the pedicles, laminae, and articular processes, whereas in flexion the forces are transmitted via the ligaments. The posterolateral inner annular fibers are subjected to maximum tensile stress in flexion, but these studies remain to be completed.

How rotation changes these forces remains conjectural,<sup>63</sup> as does the influence of fatigue.<sup>64,65</sup> Merely to maintain a flexed (stooped) position requires isometric force exceeding 66% of body weight,<sup>66</sup> yet studies of persons with and without low back pain did not demonstrate differences between them in measurable trunk strength. These findings refute the value of determining back strength as a predictor of low back pain.<sup>17,31</sup>

As the trunk flexors are very important in trunk stabilization, it is important to realize that the flexors fatigue faster than the extensors.<sup>16</sup> The dynamic endurance of the trunk muscles remains unknown and is the basis for much research. The anatomical alignment of the trunk muscles is such that no pure movement can be generated,<sup>67,68</sup> thus the integration of the central nervous system needs to be correlated in any study of dysfunction and failure.

As fatigue ensues, there is diminished control and coordination, thus exercises and strengthening, and force and endurance, have some validity in rehabilitation of patients with low back pain.



## MUSCLES IN SPINAL FUNCTION

Many studies have been directed at evaluating the isometric and concentric contraction of the low back muscles, but few have elucidated the effect and documentation of eccentric muscle contraction.<sup>69-73</sup> In the opinion of the author, the eccentric contraction of the extensor spinae muscles is predominant in most back activities of bending and lifting and should be addressed.

The position of the patient during testing has also varied from prone, supine, sitting, and standing with and without pelvic immobilization to exclude the forces of the lower extremities. With any of these constraints, a true evaluation has been difficult.

In testing for eccentric versus concentric and isometric strength of patients with or without low back pain, the same findings were apparent: there was discrepancy between flexors and extensors, but it was not significant between symptomatic and asymptomatic subjects. Eccentric contractions were considered to be conducive to muscular damage during testing even with velocity controlled.

The muscle mass being evaluated in most of these studies has been the erector spinae muscles, which traditionally arise from the lumbosacral region to insert into lumbar and thoracic transverse processes and the ribs. Their actions have been those of extension and, for the more lateral fibers, lateral flexion.<sup>74</sup>

More recently the erector mass has been depicted as comprising four divisions: longissimus thoracis pars thoracis, iliocostalis lumborum pars thoracis, longissimus thoracis pars lumborum, and iliocostalis lumborum pars lumborum. The two thoracic divisions arise from the transverse processes and ribs, forming the erector spinae aponeurosis in the lumbar region. The two lumbar divisions arise from the transverse processes of the lumbar vertebrae and insert into the ilium. These four divisions are considered to have different actions by virtue of their attachments<sup>69</sup> (Fig. 7-1 and see also Fig. 1-32).

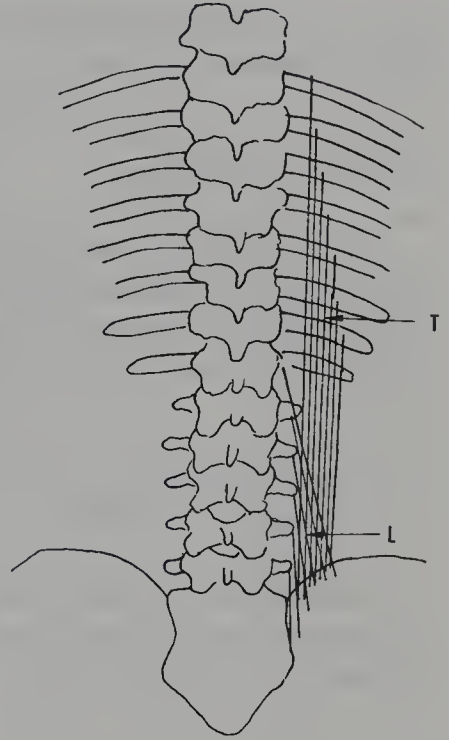
As the sites of attachment do not vary regardless of size of vertebrae or length of the fascicle, their action is constant. Their specific action can be modeled and thus computed. Their action is across numerous sections of the spinal column, as is the action of the multifidus, and it varies, as do the changes in lumbar lordosis or kyphosis.

How the erector spinae muscles function as a subsystem in the spinal stability system needs some clarification. When the trunk is flexed, the erector spinae muscles elongate and shorten in re-extension. They also exert compressive forces upon the functional units.

The configuration, direction, and angulation of the fibrils differ in the erect posture and in flexion.<sup>72,76</sup> In the erect posture, the angulation of the fibrils varies according to the degree of lordosis and the distance



Figure 7-1. Attachments of the lumbar erector spinae muscles. The attachments and insertions of the erector spinae muscles are depicted with *T* being the thoracis group and *L* the lumborum group. By evaluating each muscle fascicle the action can be determined. There are four divisions: longissimus thoracis pars thoracic, iliocostalis lumborum pars thoracis, longissimus thoracis pars lumborum, and iliocostalis lumborum pars lumborum. The first two (thoracis) arise from the thoracic transverse processes and ribs, and the latter two (lumborum) arise from the transverse processes of the lumbar vertebrae.



from the axis of rotation as well as in each functional unit. In flexion, this alignment changes with every degree of angulation (flexion) of each functional unit (see Fig. 1-24), thus it changes as the spine progressively flexes.

In flexion, there is a significant elongation of the erector spinae fibrils and changes in their orientation to the spine. As the multifidus and the iliocostalis-longissimus crisscross during flexion, they vary in their alignments and essentially do not change in their torque or compressive forces. The elongation of the fibrils (15% to 59%) reduces their active tension but increases their passive tension.

In the erect posture, the fascicles of the multifidus are oriented in a dorsocaudal direction but become ventrocaudal in flexion, whereas the fascicles of the longissimus merely align more to the longitudinal axis of the spine.

## Quadratus Lumborum Muscle

The quadratus lumborum muscle is a sagittal muscle that originates and inserts from the 12th rib, the iliac crest, and the transverse processes of the lumbar vertebrae (see Fig. 1-26). This muscle is consid-

ered to be a lateral trunk flexor and its upper longitudinal fibers essentially respiratory muscles.

## Multifidus Muscles

The multifidus muscles are so termed in the plural as they are segmental in structure. They stem from the laminae of the lumbar vertebrae and the spinous processes attaching to the sacrum. They are short muscles that act in a sagittal plane: flexing and extending the spine.

## Intersegmental Muscles

The short intersegmental muscles (see Fig. 1–25), the interspinalis and intertransversalis, are too short to exert any significant extensor strength and probably function as rotators and lateral flexors.

## Thoracolumbar Fascia

The thoracolumbar fascia encloses all the muscles of the erector spinae and the quadratus lumborum muscles. It passively plays a major role in the fibroelastic support of the spine both in flexion and re-extension. It exerts pull in conjunction with the erector spinae muscles, which can paradoxically be termed *active-passive pull* (see Fig. 1–47).

## Muscle Pain Mechanism

Muscle pain has been a recognized factor in low back pain. The mechanism is an increase in activity of the nociceptors within the muscle bellies by substances such as bradykinin.<sup>77</sup> Normal muscular activity does not activate these nociceptors.

The location of these free nerve endings is in the wall of arterioles and connective tissue of muscle.<sup>78</sup> These free nerve endings are almost completely ensheathed by Schwann cells, with only small portions uncovered and exposed to the interstitial fluid.<sup>79</sup> Their exact morphology is unclear.

Neuropeptides are considered as the substances that stimulate the nociceptor sites. No specific neuropeptide has been identified as the type that irritates muscle endings, but substance P is considered most likely.<sup>80</sup> Their peptide patterns have been similar to those of cutaneous

and visceral organs.<sup>81</sup> Many of these neuropeptides are found not only in nociceptive units but also within the spindles innervation, so the conclusion is that many of these neuropeptides are found in thin myelinated and nonmyelinated muscle afferents irrespective of their sensory function.<sup>82</sup>

These substance P (SP) neuropeptides are released not only from the spinal terminals of the afferent fibers but also from the receptive endings in the periphery. They have a strong vascular action, thus they affect the environment after their release. The bulk of the SP released in the dorsal ganglion is transported to the peripheral nerve ending<sup>83</sup> and is considered to initiate neurogenic inflammation.<sup>84</sup>

The exact mechanism by which these neuropeptides cause muscle pain is unclear. The probability exists that they cause a vasodilation into the muscle that is firmly enclosed within its fascia and increase the intramuscular pressure, resulting in pain.

The mechanical forces that act on the muscles, exciting their nociceptors, are considered to be from breaks in muscle fibers and extravasation within the muscle belly as well as the liberation of neuropeptides.

A muscle that is forced to perform physical work of unaccustomed intensity or duration undergoes histological alterations, including necrosis. This is especially true after eccentric contraction.<sup>85-87</sup> As has been stated, eccentric contraction induces trauma as the external forces acting on the muscle are greater than those produced by the muscle.<sup>88</sup> This is assumed to occur as there are fewer muscle fibers acting during eccentric contraction than in positive work of similar intensity and the mechanical elongation stress on the Z bands of the sarcomeres causes damage.<sup>89</sup>

Ischemia of the muscle has been postulated as a major cause of muscle pain. Merely interfering with the circulation of muscle does not cause pain,<sup>90</sup> but contraction of that muscle during ischemia does. What the mechanism is remains unclear. During ischemia, a plasma protein<sup>90</sup> (BKN) is released that allegedly irritates the nociceptor endings,<sup>91</sup> but that also remains unconfirmed.

Increased muscle tone as a factor in pain also remains unclear albeit being considered as pertinent. Muscle tone remains undefined as being a neuromuscular activity, as a relaxed muscle is electromyographically silent.<sup>92,93</sup> Tone has been equated as the result of a viscoelastic force such as osmotic pressure from fluids, elastic tonus within the connective tissues, and pressure within fascial compartments rather than a neuromuscular tonus. Increased muscle tone has clinically been determined from resistance to passive movement.

Pain that results from spasm or cramps remains unclear, as the intramuscular pressure has not been found to be significantly elevated.<sup>94</sup> The neural mechanism postulated as occurring is that the affer-

ent nociceptive impulses synapse with the  $\alpha$ -motoneurons at the cord level<sup>95-99</sup> (see Fig. 4-2).

A recent review of trunk strength and lifting strength measurement has summarized the current acceptance of these measures<sup>100</sup> but current traditional measures used for objective evaluation of low back disorders (LBDs) have been unable to provide meaningful, reproducible quantitative parameters to accurately classify or monitor LBDs.

Physicians attempt to classify LBDs according to pathoanatomic sources of LBD but these measures result in a precise diagnosis in less than 15% of patients with LBD.<sup>101</sup> It has been estimated that over 25% of healthy asymptomatic individuals have image-based evidence of abnormal disk herniation.

The Quebec Study<sup>1</sup> revealed that structural abnormalities are not always identifiable in LBDs and that LBDs were time dependent. Objective functional assessment systems have been explored in which measurements of the amount of force or strength the patient is willing to generate under isometric, isokinetic, and isodynamic conditions can be compared with those generated by healthy (asymptomatic) people. These measurements usually require maximum voluntary force exerted against a set resistance, usually in one plane of motion and that it is influenced by pain tolerance, which is highly variable.

Most of these tests give variable results and are not precisely reproducible in similar tests. These strength testing measures externally load the trunk in a manner that does not reflect learned patterns or coordinated neuromuscular control systems nor do they replicate actual work situations. Most studies were computed to equate the activity with compressive forces on the spine and largely ignored shear and rotational forces or acceleration during complex lifting tasks.<sup>102</sup>

Epidemiologic evidence emphasizes that twisting and symmetrical trunk motion that involve shear and rotation are related to the increase in LBDs.<sup>103-106</sup> In vitro studies have shown that spinal segments have lower capacity to withstand compression forces that are imposed simultaneously with torsional and shearing forces.<sup>107,108</sup> Cyclic rotation torsional loads also increase compressive and shearing forces.<sup>109</sup>

To date, most systems analyses study linear forces and not the actual complex spinal motions: simultaneous flexion, extension, lateral flexion, and rotation, which oversimplify the neuromuscular system.

In the healthy neuromuscular system there is a constant instantaneous interplay between the agonist and antagonist muscle systems that conform to a pattern of three-dimensional forces with a controlled acceleration. In a simple linear motion there are fewer muscles activated than are activated in complex three-dimensional motion. In a three-dimensional system the involved numerous muscles are in a constant state of flux at various points in the motion. At various points



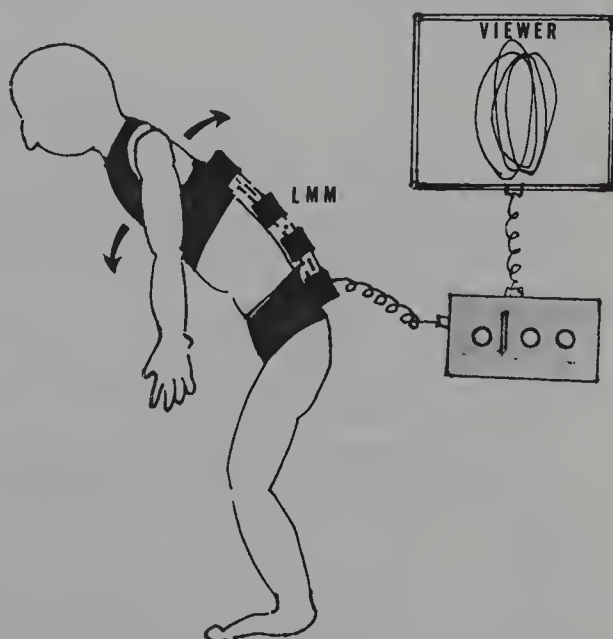
during the motion there are many muscles cocontracting rather than reciprocal action-relaxation. EMG evaluations are thus confusing. Linear studies misrepresent shear and torsional forces because all muscles are not constantly being represented.

Marras and Wongsam<sup>18</sup> developed a system that measures trunk-unloaded free dynamic motion patterns in normal and vocational situations. These studies measured velocity in three-dimensional measures of motion that have been found to occur in everyday activities. Single motor units studied by EMG have been enhanced by the concept of encruitment and coactivation of muscle units,<sup>110</sup> which occurs during a complex action such as the three-dimensional activities of the spine.

The biomechanical studies of Marras, et al.<sup>106</sup> used equipment they developed called *lumbar motion monitor* (LMM) (Fig. 7-2), which is an externally applied triaxial electrogoniometer that assesses instantaneous position of the lumbar spine in a three-dimensional space.<sup>111</sup> It tracks position, velocity, and acceleration by recording via an analogue-to-digital converter and a portable microcomputer. The repeated dots are lined forming a reportable curve on a computer disk.

The relationship of correlation of faulty motion to specific tissue sites of pain and impairment have as yet not been confirmed but the faulty motion indicates that motion is a learned or cognitive process and

Figure 7-2. Lumbar motion monitor. This externally applied piece of equipment with a chest portion and a pelvic portion is connected with a triaxial electrogoniometer. The path of trunk motion is viewed on a monitor that can be recorded on computer disk. The person can flex, extend, rotate, and perform any combination of these movements. The range of motion as well as the velocity and acceleration is recorded over time. (Equipment was developed by Biodynamics Laboratory and Statistics and the Division of Orthopedic Surgery, Ohio State University, Columbus, Ohio.)





resultant pathology (LBDs) may reflect changes in coordinated recruitment of the neuromuscular system with resultant tissue injury. Faulty body mechanics that have been impugned for many decades is becoming clarified.

Free dynamic measurement rather than strength-based loaded studies are more subtle and informative as they simulate actual daily activities that stress-loaded studies do not.

Cognitive processes affect biomechanical processes and explain learned behaviors influenced by pain sensitivities.<sup>112,113</sup> The deviations from pain-inhibition fear avoidance,<sup>114</sup> psychological distress,<sup>115</sup> and illness behavior<sup>116</sup> are revealed in unloaded acceleration three-dimensional studies.

This model (LMM) may ultimately reveal patterns for specific LBDs as well as understanding the ergonomics in the workplace. Once standardized it may set a base of neuromuscular function in the patient with LBD and offer a basis for monitoring progress from a therapeutic approach.

If the faulty neuromuscular activity is understood the resultant pathoanatomic sequelae will become more evident and therapeutic approaches will be monitored physiologically in a recordable manner.

Many of these studies have been implemented in patients with chronic low back pain with well established patterns. It remains to be seen if similar studies will be effective in evaluating acute low back disorders when the developing neuromuscular patterns are in their developmental stages.

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## CHAPTER 8

# Neurohormonal Aspects of Discogenic Disease

The mechanical aspects of discogenic disease are numerous and are being clarified repeatedly both experimentally and clinically.<sup>1</sup> What predisposes certain people to be prone to adverse effects resulting in disk disease from these mechanical factors remains unclear and even unexplored.

The tissues that react with pain when injured have been elucidated. The chemicals liberated from disk injury allegedly resulting in radiculopathy have been isolated as phospholipids a and b,<sup>2</sup> kinins, substance P, and many others being discovered daily.

There are mechanical factors involved in discogenic disease and there are psychological factors involved in resultant pain, but what is the relationship between them? Even fear-avoidance beliefs are now clearly considered in evaluating the disability.<sup>3</sup> The cause and effect of psychological factors regarding low back pain have been in conflict,<sup>4</sup> yet all clinicians are convinced of a relationship.<sup>5-9</sup> Barr<sup>10</sup> stated, "Low back and sciatic pain signify a state of stimulation of the central cortex and are not an affliction of the back. It is important to study and treat the patient and not just the back."

The chemical and pathophysiological factors of discogenic disease have presented biomechanical aspects. Increased disk pressure has been demonstrated to cause low back pain.<sup>11</sup> The initial changes within the disk occurred in the nucleus, with annular tears beginning centrally and proceeding outward.

The fluid content of the disk was shown to result from imbibition<sup>12</sup> with a sodium transfer.<sup>13</sup> Besides mechanical factors, the change in the water content of the disk remained unclear.

The psychological factors involved in disk disease became noted but not clarified<sup>14-16</sup> other than in the treatment of pain<sup>17</sup> to avoid chronicity. A recent study<sup>18</sup> has assessed the psychopathology in chronic and acute low back pain patients with Structured Clinical Interview for DSM-III-R (SCID)<sup>19,20</sup> and found a much higher base rate of psychopathology in patients who develop chronic low back pain. The psychopathology was major depression plus substance abuse and personality disorders.<sup>21-23</sup> Many had a premorbid psychopathology.<sup>18</sup> Whether these patients with low back pain had discogenic disease has not been equated, but it seems that many probably did.

In a current provocative dissertation Sapolsky<sup>24</sup> has equated stress with the aging brain and the mechanism of neuron death. He postulates that various stressors stimulate endocrine response dominated by adrenal secretion of glucocorticoids (Fig. 8-1). These glucocorticoids are a natural phenomenon, yet when they are excessive or prolonged, they initiate a failure of feedback inhibition, with ultimate hippocampal loss of receptors. Sapolsky develops this "cascade" concept, alleging that uncontrolled glucocorticoid secretion ultimately leads to neuronal death or impairment.

In his text, he states that major depressions have been associated for 30 years with excessive quantities of cortisol and with insensitivity to glucocorticoid feedback inhibition.<sup>25</sup> This may be due to or aggravated by hippocampal dysfunction.<sup>24</sup> The hypercortisolism of depression involves hypersecretion by the brain, which desensitizes the pituitary, causing hypersensitivity to adrenocorticotrophic hormone (ACTH) by the adrenal glands. With depletion of hippocampal receptors, this may explain the hypercortisolism of depression.

With this hypersecretion of cortisols, the effect upon the collagen within the intervertebral disks can be assumed albeit unconfirmed. It is an alluring concept that depression precedes disk degeneration rather than following it.

With sodium transfer being involved in disk hydration, what affects sodium has been studied in numerous entities, some of which are related to disk pathology. Depression has been demonstrated to be related to sodium metabolism,<sup>26</sup> and probably also related to corticosteroid metabolism,<sup>27</sup> which is related to fear, anxiety, and depression.<sup>28</sup> Psychological distress and emotional arousal have been known to increase steroid secretion, but why depression seems prominent in disk disease has not been answered.<sup>29</sup>

A sequence of events may cause elevation of intradiskal pressure that may cause low back pain. If repeated or persistent, this elevated internal pressure plus external muscular tension may invoke annular tearing.<sup>30,31</sup> Degradation in protein polysaccharides causes increased fluid retention, with further increase in intradiskal pressure. A new

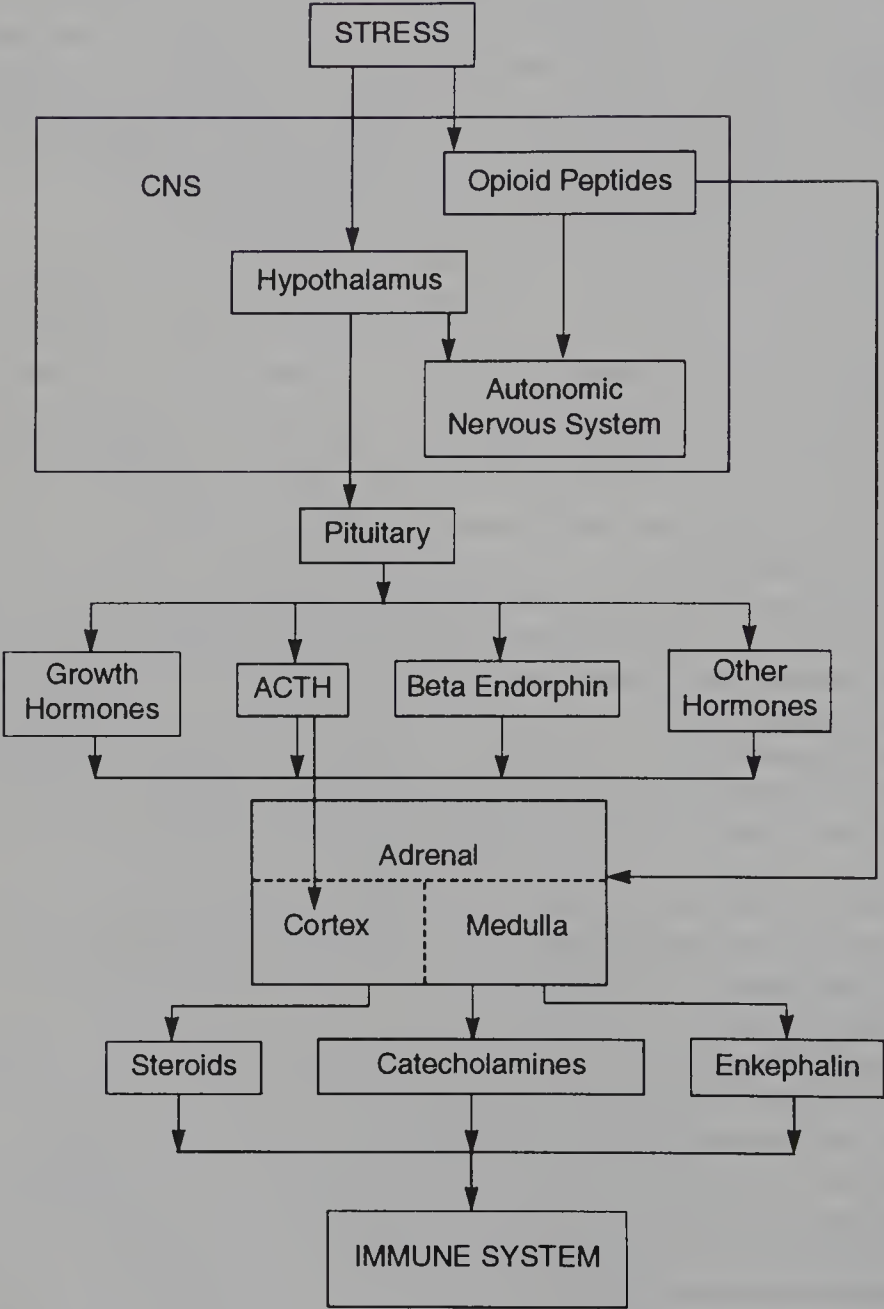


Figure 8-1. Sequence of neurohormonal aspects of stress. (Modified from Shavity, I, et al: J Immunol 135:836S, 1985.)

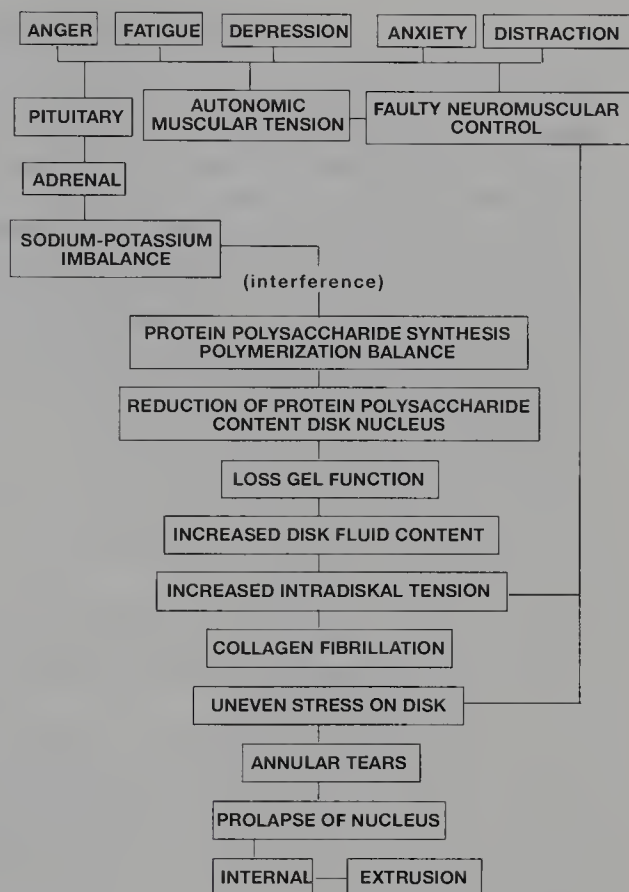


Figure 8-2. Psychophysiological algorithm of stresses on the intervertebral disk.

synthesis of protein polysaccharides of a lower level ensues, which has an increased ability to imbibe fluid (Fig. 8-2).

Collagen changes occur during this sequence. Polysaccharide proteins have a plasticizing effect on the collagen fibrils, coating them as they glide (sheet on sheet) in their function. This plasticity is diminished and lost with degradation of the protein polysaccharides.

The vertebral intradiskal pressure varies with its hydration. Under a load of 100 KP, the nucleus loses 8% of its water content and the annulus 11%, with the greatest loss in the dorsal aspect. Under pressure, the sodium and potassium increase more in the nucleus than in the annulus.<sup>32</sup>

The external neuromuscular tension imposed upon the disk<sup>33</sup> is increased by emotional tension.<sup>26</sup> As emotional tension increases, there is an increase in recorded muscular tension.<sup>34</sup>

Regarding a relationship of emotions and intradiskal pressure, herniation, and ultimately degeneration, a psychophysiological explanation is advanced.



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## CHAPTER 9

# Disability Evaluation

The complaint of low back pain from both personal injury and industrial accidents has led to enormous expenses.<sup>1</sup> The impairment associated with the clinical evaluation of the low back<sup>2,3</sup> must also be equated with disability, wherein lies a major controversy. A provocative paper by Wolfe<sup>4</sup> has attempted to delineate impairment from disability and the role played in its determination by the medical examiner.

The term *disability* remains unclear and may represent *functional disability* as related to ability to engage in the activities of daily living as well as gainful employment. Functional disability must scale qualitatively and quantitatively the degree of disability. In this aspect disability must also evaluate the impact of psychological dysfunction as related to anxiety, depression, and even psychosis.

Work disability has been included in the definition but again must be clarified as impairment to perform all or merely some of the job requirements. The World Health Organization<sup>5</sup> (WHO) presented a model of disability in which *disease* leads to *impairment*, which may lead to *disability*, which in turn may lead to *handicap*. Impairment is relatively objective, whereas disability is subjective. Handicap is the social sequela of disability. Work disability is, in fact, handicap within the WHO system<sup>4</sup> (Table 9-1).

Patients may have significant functional disability yet not be work disabled, depending on the demands of the employment and the precise type and extent of the impairment. Disability therefore relates to the demands of the job and is modified by education, training, interest, determination, support from peer group, and many other factors.

In today's society physicians are asked to assess a patient's ability to work. Employers, attorneys, compensation agencies, and insurance companies ask physicians whether the patient can work or is limited in

Table 9–1. WORLD HEALTH ORGANIZTION MODEL OF DISABILITY

Category	Definition
Impairment	Pain Psychological dysfunction or distress Muscle dysfunction (possible)
Disability	Functional disability: Inability or limited ability to perform ADL or IADL tasks Psychological disability: Limitations in personal and interpersonal activities
Handicap	Work disability: Inability or limited ability to be employed or to do work

From Wolfe, F: Disability and the dimensions of distress in fibromyalgia. J Musculo-skeletal Pain 1:67, 1993, with permission.

ADL = activities of daily living; IADL = instrumental activities of daily living.

the ability to work. Physicians are actually requested to estimate the percentage of disability related to the impairment and to the assumed causation. Wolfe<sup>4</sup> claims that the physician actually “knows less about the patient’s ability to work than than does the patient himself.”

According to WHO, the physician can ascertain the relationship of disease (injury) to impairment and also to functional disability in spite of the fact that the etiology of the disease and its subsequent impairment are unclear, as is so apparent in low back pain syndrome.

With advancing knowledge of pain and its mechanisms,<sup>6</sup> it is accepted that peripheral pain induces central changes in the nervous system that are related to genetic, familial, and psychological factors.

When impairment is determined according to WHO standards, the exact roles and relationships of pain, psychological reaction, and structural tissue changes of the injured anatomical part cannot be determined.

The disability process and evaluation depicted by Wolfe<sup>4</sup> in fibromyalgia can also be envisioned in low back pain. The physician is involved by numerous parties:

1. A patient being treated requests a work disability status.
2. A patient seeks a physician to establish a disability status.
3. An attorney requests a disability status.
4. An opposing attorney or insurance company requests confirmation (or denial) of work impairment status.
5. Health insurance or health care providers request fulfillment of disability status forms.

6. Medical records of health care providers are requested or subpoenaed.

The practitioner is requested to ascertain the relationship between the injury, the resultant impairment, and the disability.<sup>7-9</sup> The physician is asked to determine whether the patient is totally or only partially disabled, how long the disability will continue, the response to allegedly appropriate treatments and whether modification of work conditions or rehabilitation will be effective. The ability of a physician to answer all of these questions in patients complaining of low back pain is highly questionable.

An answer is currently adversarial, with the physician being swarmed by the patient, spouses, family, attorneys, insurance companies, compensation agencies, or often other physicians.

An algorithm was proposed by Wiesel et al.<sup>10</sup> (Fig. 9-1). They stated that "there is no standardized protocol for diagnosis and treatment of low back in industry. Diagnostic criteria and treatment regimens are haphazard and may vary from patient to patient in the same doctor's office. The outcome for the patient depends on legal implications, the physician's mood, and the patient's own motivation."

They proposed the following protocol and conclusions:

1. Good medicine leads to cost savings in treating industrial low back pain.
2. Use of a standardized medical approach and nomenclature is necessary, and practical, for consistent care.
3. A good record-keeping system is essential to perform useful medical analyses for identifying scientific problems.
4. Unbiased medical surveillance leads to changes in behavior of both treating physicians and patients.
5. The outcome for most low back pain patients in industry is not grim, as previously perceived, if their medical management is approached in an organized manner.

This protocol is idealistic and has yet to be implemented as many of the factors remain unclear. These factors within the algorithm are also unclear.

The goal of evaluation and treatment of the low back injured patient is prompt return to work, as total relief of pain is not always achieved. A second goal is that diagnosis and treatment must be performed at an acceptable cost to society. This leads to a third goal, which relates to the first two: avoiding unnecessary and ineffectual surgery. All will be addressed as the problems are monumental and as yet unsolved. The remainder of this text will be an evaluation of segments of



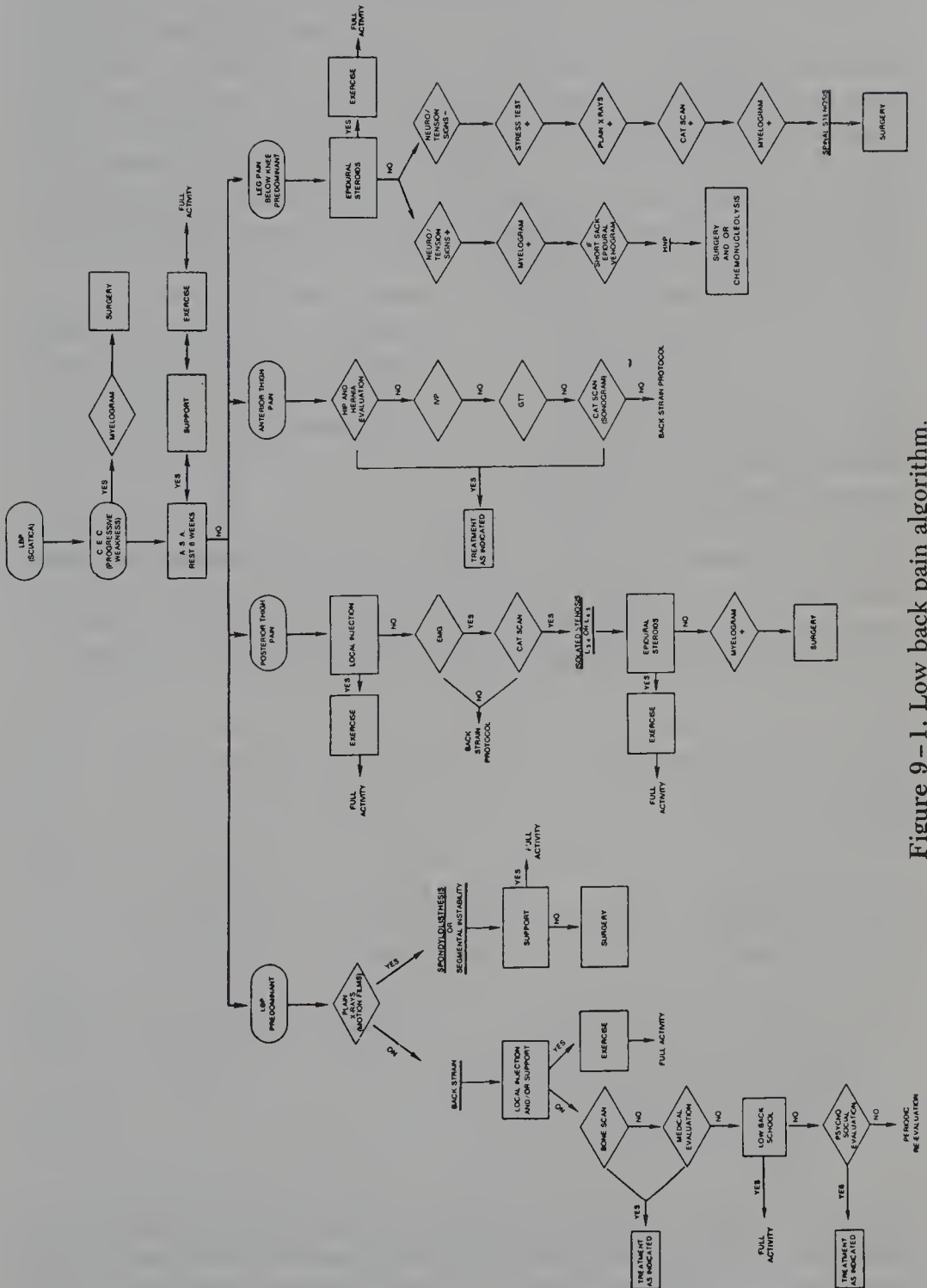


Figure 9-1. Low back pain algorithm.

the algorithm (Fig. 9–2), which generally is accepted by most practitioners as a guideline for evaluation and treatment.

Patient education is germane to these goals, as continued activities after injury are frequently painful and interpreted by the patient as injurious. Physicians are also influenced by this concept, as labels are immediately placed on the condition, which is poorly understood by the physician and totally misinterpreted by the patient. Meaningless treatment ensues that prolongs the disability in the absence of organic impairment. Useless tests and x-rays are incurred, and the condition is labeled with innocuous yet threatening terms such as *discogenic disease*, *degenerative arthritis*, and so on. An addition to the algorithm in Figure 9–2 could be “education” (added between 7 and 8).

Conservative treatment that is considered definitive is too often useless and ineffectual (7, 8, and 9 in Fig. 9–2). It is often prolonged, leading to surgical intervention. Surgery is involved to “remove” the pain, but this does not occur. Surgical intervention (6 in Fig. 9–2) should be reserved for the removal of organic pathology, which is the cause of pain and impairment.

Precise diagnostic studies are also misused, abused, and misinterpreted. Too often, pathology noted on these studies leads to surgical intervention, whereas the pathology may not be the basis of pain or impairment. Studies should be used only when there is clinical evidence of impairment and the studies thus used for confirmation or denial.

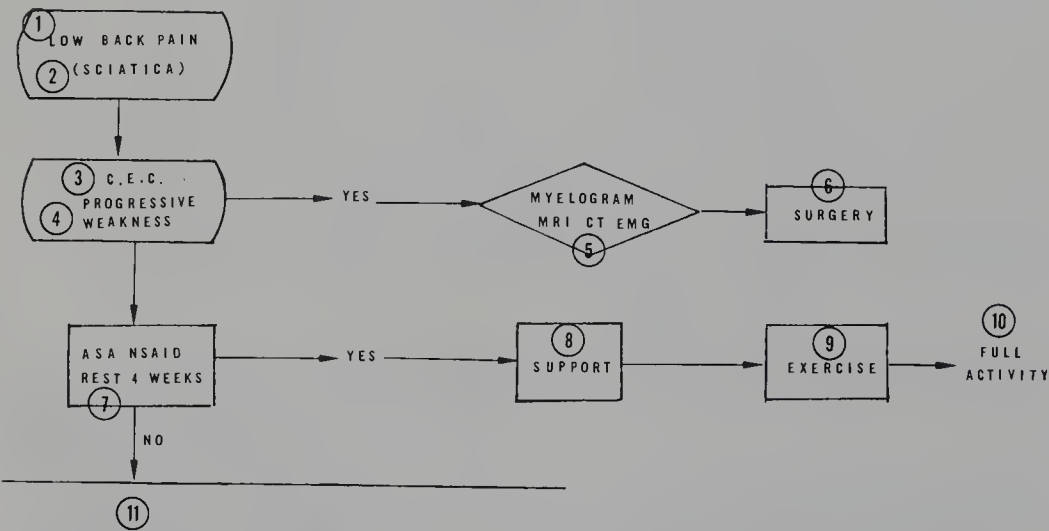


Figure 9–2. Algorithm of treatment protocols in low back pain syndromes. (1) Indicates merely low back pain; (2) low back pain with sciatic radiation; (3) caudal equinal compression (CEC); (4) myotomal weakness; (5) diagnostic studies; (6) surgical intervention; (7) nonsteroidal anti-inflammatory medications and rest; (8) support such as a corset; (9) exercise; (10) return to full activity.

The algorithm proposed divides the problem between low back symptoms with or without leg symptoms and involvement of bladder function. The pathology which merely causes low back symptoms differs from pathology that invokes leg and bladder symptoms.<sup>11</sup> The presence of bladder involvement termed cauda equina syndrome<sup>12</sup> (EEC in 3 of Fig. 9–2) constitutes the only surgical emergency other than the condition of rapid progressive true motor weakness.<sup>13</sup> Fortunately the condition of equinal syndrome is rare, but it must be immediately recognized, as it mandates surgical intervention.

The remainder of the algorithm devoted to predominant low back pain without leg symptoms is where questions arise. The usual condition is termed benign if there is no structural pathology such as spondylolisthesis. Segmental *instability* has been propounded, but this term or entity still awaits clear definition.

Low back pain (1 in Fig. 9–2) is where toxonomy remains obscure. The diagnostic definition presents confusion as to etiology, essential symptomatology, confirmatory physical examination, indicated therapy, and prognosis. To computerize the diagnosis as to statistical evaluation and outcomes assessment, the failure of an accepted standardization of toxonomy presents a major obstacle. The ensuing algorithm is meaningless without this term (*diagnosis*) being clarified.

Terms such as *low back strain/sprain*, *discogenic syndrome*, *minor subluxation*, *facet syndrome*, and *degenerative disk disease* are among the numerous labels attached to symptomatic low back pain patients. Such labels are meaningless in that they fail to implicate the true mechanism of how and why the patient sustained impairment or pain from an activity. These labels fail to implicate the precise anatomical structure that causes nociception. The psychological aspects of the incidence (injury) are noted at a later date and, in many cases, are accusatory, incidental, and improperly used.

As a summary statement, the violation of proper body mechanics from external forces or internal functions that cause irritation of a structure within the functional unit is the cause of pain and impairment. Once this is ascertained from an appropriate history followed by a meaningful physical examination revealing the tissues affected, the symptoms become apparent and the impairment discernible. Further implication of this etiological factor becomes meaningless if the specific irritated tissues are not addressed and the symptoms clarified.

In today's medical model, the symptoms rather than the impaired function are addressed. The *disability* is the condition that is assumed to result from the *impairment*, and the physician compounds the problem by invoking the concept of *illness* rather than *condition*, and meaningless studies ensue, inappropriate treatment protocols are invoked, and disability becomes sensory rather than functional.

The sites of tissue nociception within the functional unit of the vertebral column have been identified and isolated. The sensation provoked by the insult termed *pain* is still unclear, although many clinical studies have proclaimed which symptoms evolve from specific tissue types. *Ache*, *throbbing*, *soreness*, and *tightness* are some of the many terms employed. *Excruciating*, *paralyzing*, *restricting*, *disabling*, *unacceptable*, and so on are used in an attempt by the patient to describe intensity. All are subjective qualification and quantification terms describing the symptoms, and the physician thereupon attempts to justify and explain these symptoms by an examination. A label (diagnosis) ensues.

The mechanisms of the low back function have been well documented, and the medical literature is extensive. The functional unit is the focus of function, with the following tissue sites of nociception being implicated<sup>14</sup>:

1. Outer annular fibers
2. Longitudinal ligaments (especially the posterior ligament)
3. The nerve root dura
4. The capsules of the facets
5. The erector spinae muscles
6. Paraspinous ligaments

The innervation of these tissues is paramount as nociception demands neural transmission via C fibers and slightly myelinated nerves to transmit sensations that will ultimately be conceived of as pain.

The mechanism by which injury occurs to one or more of these nociceptive tissues is invoked by the history. The involved tissue is determined by the examination. The resultant pain and impairment are determined by the involved tissue being momentarily removed from normal function.

How the spine functions, therefore, must be precisely considered and the mechanism causing impairment understood. The resultant inflamed tissue becomes apparent.

The usual injury occurs from improper bending and/or lifting. Because of this implication, normal flexion and re-extension combined with lifting will be carefully evaluated in today's knowledge, then improper bending, re-extending, and/or lifting will be analyzed. The tissue reaction causing pain and impairment will become apparent. Subsequent chapters will depict the appropriate history, and details of examination will evolve.

The ability of physicians to determine disability or even impairment is very poor.<sup>15</sup> Review of reports over 10 years of independent medical examiners in California consistently showed great differences in the



estimates of disability.<sup>16</sup> The International Association of Industrial Accidents Boards and Commissions<sup>17</sup> agreed on the inconsistency of disability evaluations.

As has been repeatedly stated in this text, the unavailability of objective signs makes a reliable conclusion difficult. The prevalence of psychological and psychosocial aspects of disability has also been difficult to evaluate and quantitate.<sup>18,19</sup> An anatomic diagnosis, albeit accurate, does not equate with symptoms and disability.<sup>20</sup>

Important data factors were discovered as missing in most disability evaluations.<sup>17</sup> There were incomplete and irrelevant histories, inconsistent physical examinations, reports of irrelevant findings, inappropriate tests, and inaccurate interpretation of these tests. Guideline factors of quantifying disability were itemized<sup>15</sup> and will be partially summarized and discussed:

1. History of back pain or leg pain, or both, resulting from an injury, with resultant interference with work for 90 days.<sup>15</sup> When such a history is elicited, the precise mechanics should be documented, such as the exact mechanism of injury and the patient's psychological status at the time of injury, whether fatigued, impatient, angry, or the like. The statement "I was hurt" should be replaced by "I hurt myself."
2. Previous episodes of low back injury with same details elicited, including the received attention and the duration of disability.
3. Requirements of the job as described by the patient as compared with the job description given by the employer.
4. History of previous spine surgery in precise details.
5. History of heavy lifting regarding manner, frequency, obstacles, and "meaning of the efforts" (patient's interpretation).
6. Radicular pain in specific detail and motor loss in precise detail. Dural signs, and so on, as discussed in Chapter 3.
7. Proper evaluation of straight leg raising and objective neurological examination.
8. Pertinent evaluation of confirmatory tests as to their relevance to present symptoms and objective findings.
9. A personality profile can be elicited by proper questioning and, if indicated from examiner's concern, be tested.<sup>21</sup>
10. The frequently used pain drawing has value if properly interpreted,<sup>22</sup> but it must be carefully used to give an objective disability status. The presence of Waddell's nonorganic physical signs must be kept in mind.<sup>23-26</sup>
11. Response to treatment must be evaluated,<sup>27</sup> but this is with the understanding that the treatment is appropriate, time is limited, and physiological. Today these criteria are difficult to state.



In the quantification of function<sup>28</sup> it is difficult to differentiate the objective from the subjective as many are influenced by pain, attention, cooperation, and interpretation of significance.

Disability evaluation remains conjectural in spite of the voluminous literature and the multitudinous numbers of criteria advocated. The interpretation by the physician also remains varied by experience, training, and motivation. The day of acquiring an objective, consistent, reproducible, and available method of quantifying disability is not yet here.

The psychological and psychosocial aspects are predominant yet also are not amenable to objective documentation. The psychological component of disability still remains in the obscure category of accusation for secondary gain with no means of differentiation.

A plea is made to ensure correct meaningful history taking, performance and interpretation of objective findings, and signs and utilization of meaningful treatment protocols.

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## CHAPTER 10

# Treatment Protocols for Low Back Pain Syndrome

Treatment protocols must relate to whether the low back pain is acute or chronic. An intermediate condition that must also be evaluated is recurrent low back pain. Prevention of recurrence also falls into the category of treatment.

As has been emphasized, pain is the predominant symptom bringing the patient to the health provider. Pain remains the focus of most care, and chronic pain the most prevalent factor of persistent impairment and disability. Before the numerous aspects of pain and its significance in relationship to disability are discussed, pain, per se, needs clarification.

Pain has undergone intensive study. In the Medline Citation Index the total number of articles devoted to *pain* was 346 to 1971, 876 in 1981, and 1645 in 1991.<sup>1</sup>

Pain is a warning signal that helps protect the body from tissue damage. Sherrington<sup>2</sup> defined pain as a psychological adjunct to a protective reflex, the purpose of which is to cause the affected tissues to be withdrawn from the potentially noxious (and injurious) stimuli.<sup>2</sup> Pain, unlike most other sensory modalities, has the essential function of survival. Pain, however, also has a psychosocial aspect that will receive significant attention.

The neurophysiology of pain is becoming increasingly recognized and verified.<sup>3</sup> Sensation of pain originates from the activation of nociceptive primary afferents by intense thermal, mechanical, or chemical stimuli. These nociceptive sites are small, free nerve endings in the numerous tissues of the body. In the low back these sites have been

**Figure 10-1.** Kneeling man perceiving pain (Descartes). The Descartes kneeling figure (1596–1650) depicts a burning sensation irritating the filaments of a nerve in the foot ascending to the brain via a single filament of that nerve. (Rene Descartes' illustration from "De l'Homme" was modified by the author.)



elucidated in the preceding chapters, as have the numerous nociceptive stimuli and their mechanisms.

The accepted neurophysiological concepts of pain transmission and interpretation have undergone many changes in recent decades, but clarification and acceptance are increasing.

The acceptance of pain as being exclusively a peripheral manifestation at its receptor sites as a result of tissue damage going directly to the brain is no longer tenable (Fig. 10-1). Originally pain was considered to be dependent solely on the intensity of the noxious stimulus, but this has been refuted.

The concept that specific nerve endings in the involved tissues were also specific in the sensation translated has also been refuted.<sup>4</sup> This concept postulated that specific nerve endings produced specific pain sensations. It is now known that there are specific nerve types that transmit sensation that will ultimately be interpreted as pain.

Normal forces on skeletal tissues, both external and internal, cause tissue deformation (see Fig. 4-8), which is reversible when these forces are removed. When excessive, these forces cause irreversible tissue damage, which ends up liberating nociceptors (see Fig. 4-6).

These nociceptors,\* pain-producing substances termed *algogens*, are released at the site of tissue injury. The algogens are numerous and

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\*Nociceptive impulses: impulses giving rise to sensations of pain. In Thomas, CL (ed): *Taber's Cyclopedic Medical Dictionary*, ed 17. F.A. Davis, Philadelphia, 1993.



are added to frequently.<sup>5-8</sup> Leukotrienes are one of the liberated algogens as are phospholipids that break down to arachidonic acid and prostaglandin E. Local tissue trauma breaks down blood platelets that release serotonin, which acts as a vasoconstrictor, causing local edema. Other algogens are histamine, substance P, somakinin, vasoactive polypeptides, and cholecystokinin, to mention a few. Many are vasodilators or vasoconstrictors as well as irritants.

In the low back this edema causes compartment compression, as the erector spinae muscles are contained within partially inflexible fascial sheaths (Fig. 10-2).

The enclosed nerves contained within their individual sheaths are compressed, causing intra-arachnoid pressure, which can cause temporary transmission inhibition and when prolonged can cause intraneural fibrosis.

Transmission of nociceptive impulses is now well accepted as being via unmyelinated C fibers and lightly myelinated A alpha fibers, which

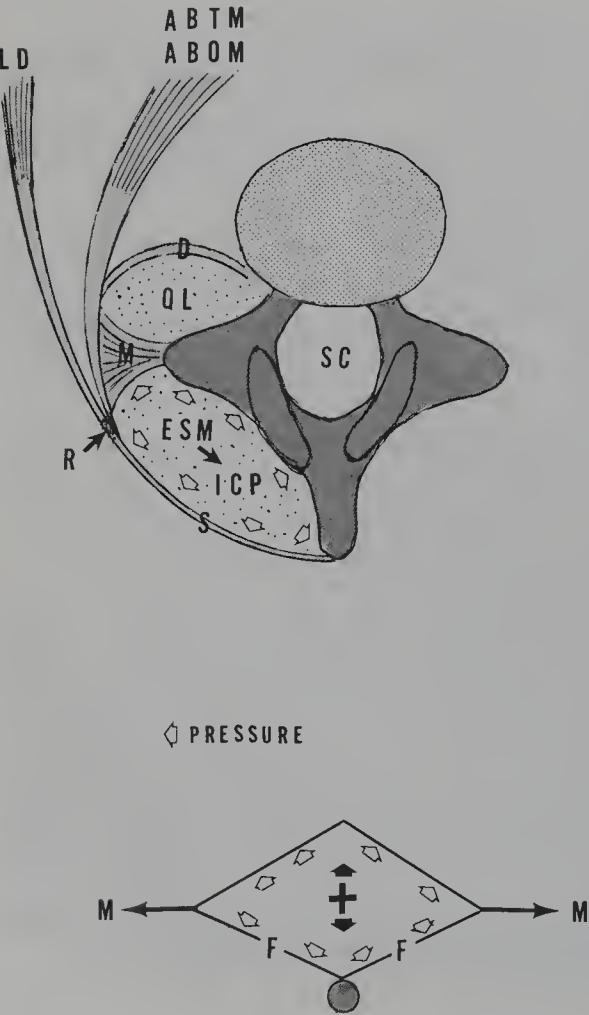


Figure 10-2. Fascial sheaths enclosing low back extensor muscles: the compartment syndrome. Schematic compartment syndrome (*bottom*) shows the contraction on the fascia (F) by the muscles (M), causing increased pressure (*small arrows*) within the compartment (+). The compartment (*top*) formed by the fascia (S) that surrounds the erecto-spinal muscles (ESM) where the intracompartmental pressure is formed (ICP). The fascia of the latissimus dorsi muscles (LD) and oblique abdominal (ABOM) and the abdominal transversus muscles (ABTM) meet to form a raphe (R). A middle fascial sheath (M) that surrounds the quadratus lumborum muscles (QL) forms the anterior wall of the compartment. The deep layer (D) of the thoracolumborum fascia encloses the quadratus lumborum muscles. The vertebra with its spinal canal (SC) is shown.



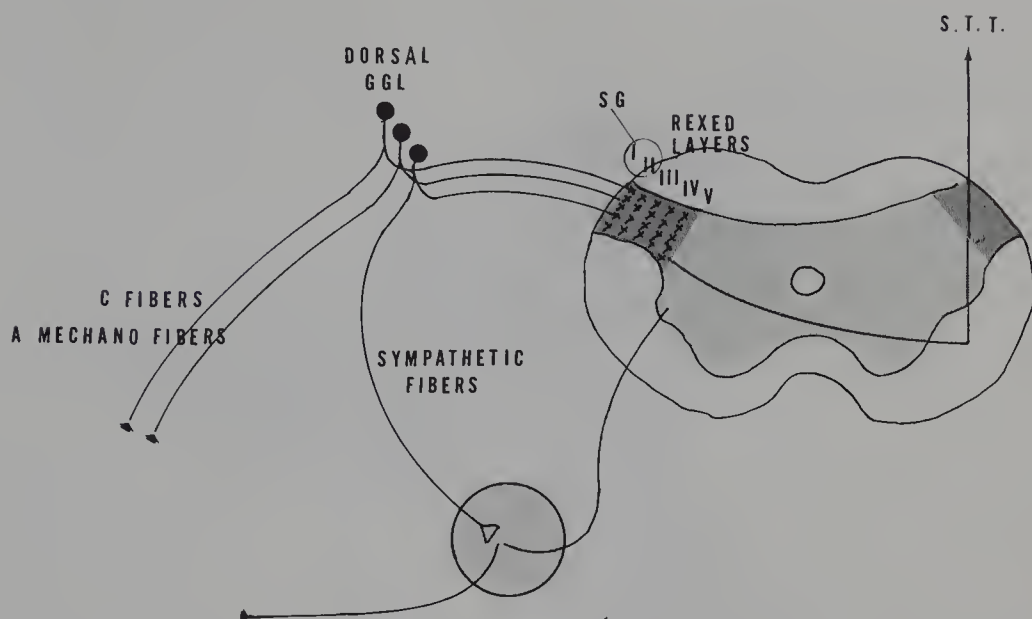


Figure 10-3. Sensory afferent fibers entering the dorsal horn of the cord. Sensory C (and A alpha fibers), mechano A fibers and sympathetic (sensory) afferent fibers enter the dorsal horn of the gray matter of the cord through the dorsal root ganglion (GGL). The dorsal horn is divided into numerous Rexed layers (I-VII). The afferent fibers enter Rexed layers I to II, termed the *substantia gelatinosa* (SG), from where they transverse the cord to ascend cephalad via the spinothalamic tracts (STT).

ascend to the dorsal root ganglia (Fig. 10-3) on their way to dorsal gray columns, with 80% of afferent nerves transmitting impulses via unmyelinated nerves as pain sensations. Unmyelinated nerves transmit impulses slowly. The large-diameter, myelinated, faster-transmitting fibers transmit stimuli such as touch, temperature, and proprioception.

Anatomists have recently demonstrated that approximately one third of all afferent small-diameter, unmyelinated C fibers enter the cord through the anterior route,<sup>9</sup> with their cell bodies probably being located in the dorsal root ganglia. This may be a neurological basis for muscle pain.

The dorsal horn is divided into laminae (rexed layers) (Fig. 10-4), into which nociceptive fibers (C and A alpha) synapse in laminae I to IV to fibers that ultimately traverse the cord to ascend the lateral spinal thalamic tracts (Fig. 10-5) to the thalamic region (Fig. 10-6).

Most peripheral nociceptive fibers terminate in laminae I to IV, although two types of fibers (fast and slow) terminate in lamina V. The A delta fibers transmit faster and carry sharp pain, whereas the C fibers are slower and carry dull, longer-lasting pain.

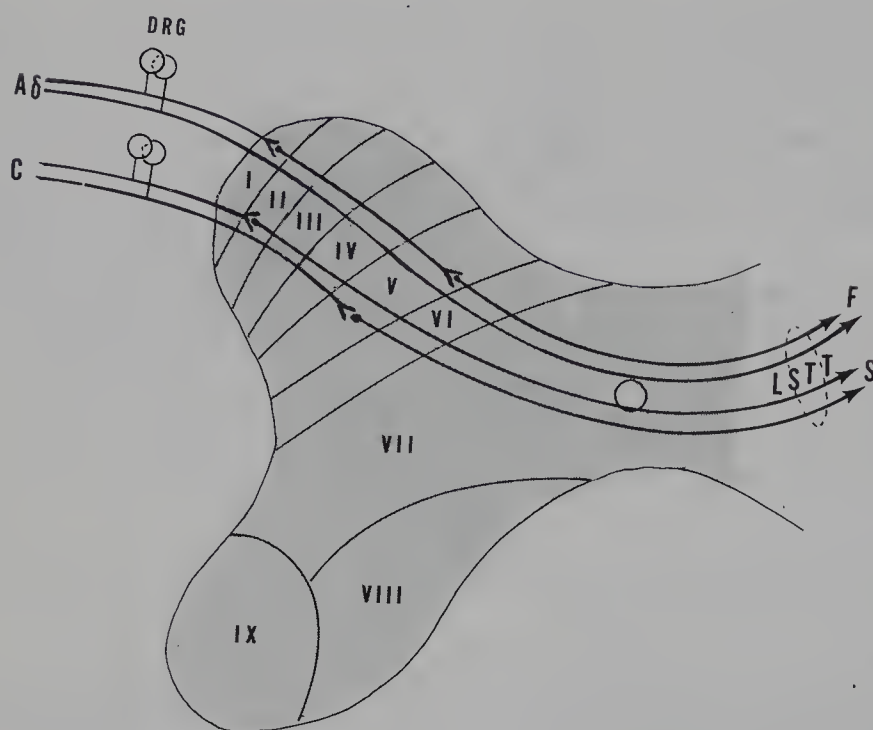


Figure 10–4. Dorsal horn Rexed layers. The C and A alpha afferent fibers enter the gray matter of the cord via the dorsal root ganglia (DRG) where they synapse in Rexed layers I, II, and V. The fibers proceed transversely to ascend cephalad in the lateral spinothalamic tracts (LSTT) as fast (F) and slow (S) impulses.

There are intrinsic factors in the central nervous system that modulate pain. Endogenous opioid substances are now considered to be synthesized by and within the nerve cells. These substances mimicking narcotic and analgesics are termed *endorphins* (*enkephalins*).

The question was raised, “Why does the brain need this pain-modulating system?”<sup>10</sup> Enkephalins act at peripheral sites in the ascending system as well as at the dorsal root ganglion, spinal cord (dorsal horn), midbrain, hypothalamus, periaqueductal gray area, and at the rostral medulla.<sup>11</sup>

The International Association for the Study of Pain (IASP) has characterized neural pain mechanisms as along five axes: (1) site of pain, (2) physiologic system, (3) temporal pattern and recurrence, (4) intensity and duration, and (5) etiology.<sup>12,13</sup> This has been directed principally to chronic pain but applies equally to acute pain that may continue into chronic pain if not interrupted.

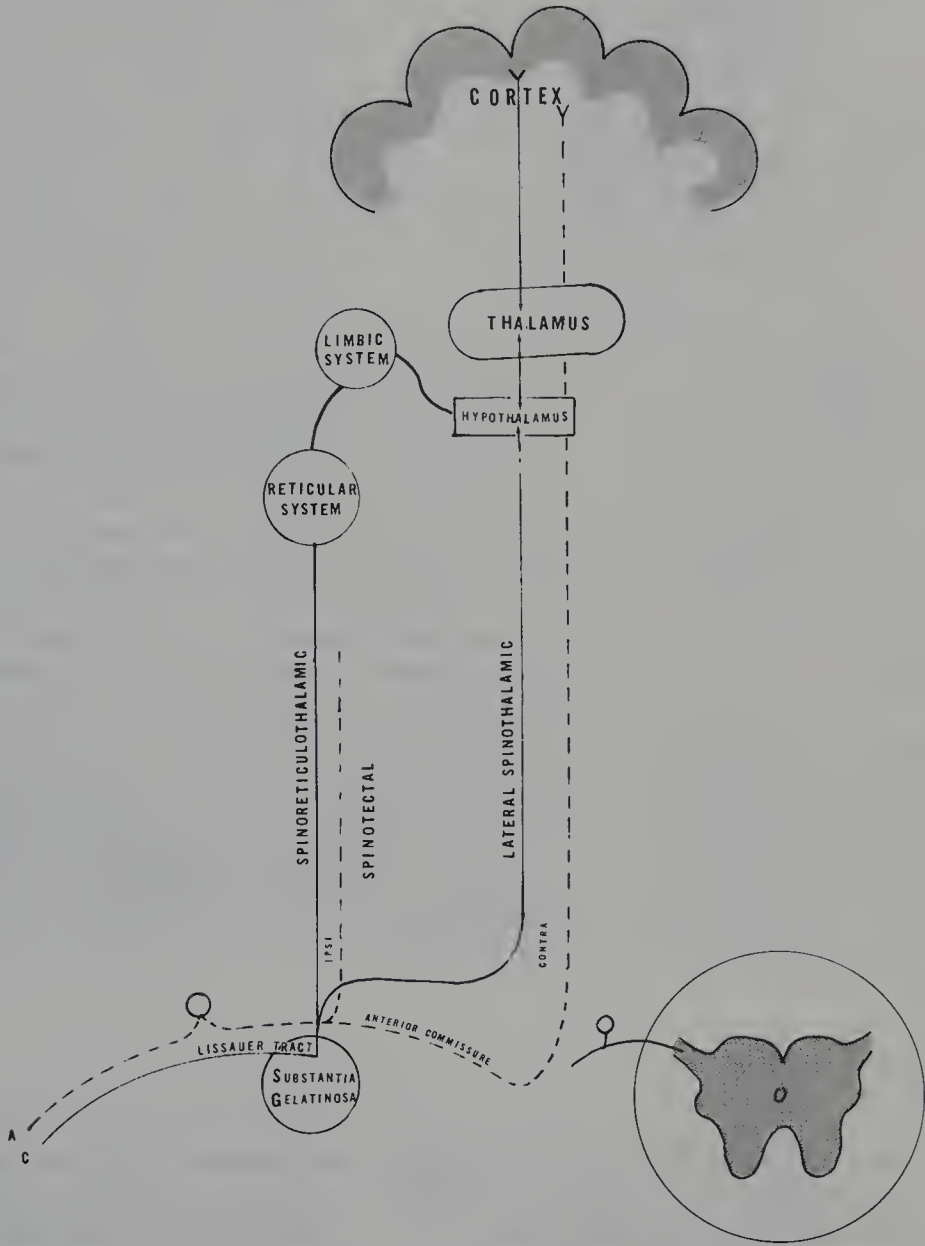


Figure 10-5. Major ascending sensory pathways of the spinal cord. The two major pathways for pain transmission are the spinothalamicocortical tracts and the spinoreticulothalamic tract. The former has spatiotemporal localization and the latter no localization but transmits emotional (limbic) and avoidance reaction. Afferent A and C fibers enter the Lissauer tract of the substantia gelatinosa (Rexed layers I and II). The pathways are labeled.

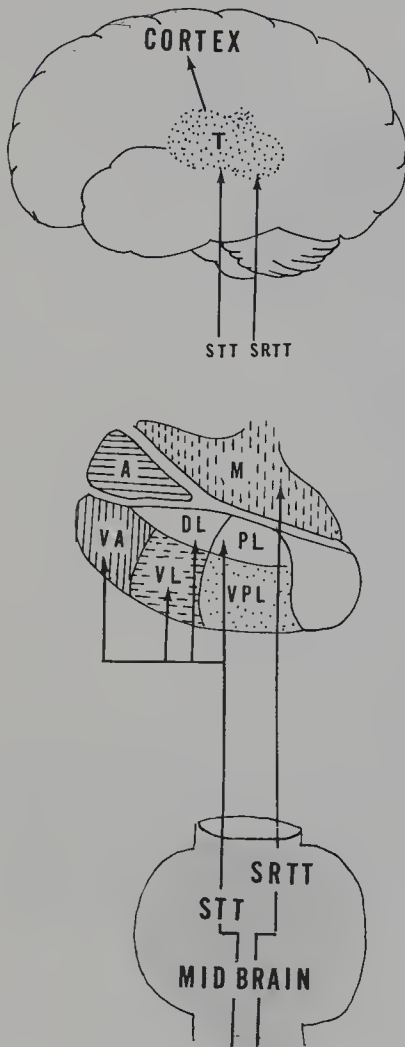
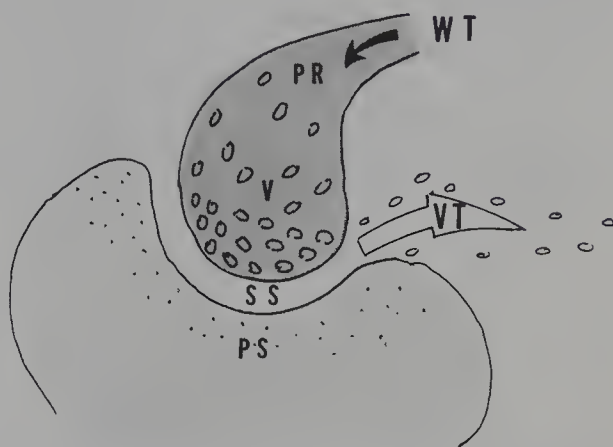


Figure 10-6. Thalamic pathways. The thalamus (T) is a large ovoid gray mass located on either side of the third ventricle. The anterior tubercle (A) is thin and lies close to the midline. The posterior portion is known as the pulvinar. The ascending tracts from the cord through the midbrain: the spinoreticulothalamic tract (SRTT) and the spinothalamic tract (STT) synapse within the thalamus (*middle*). They go directly to the anterior (A), medial (M), ventro-anterior (VA), ventrolateral (VL), dorsolateral (DL), posterolateral (PL), and ventroposteriolateral (VPL). From the thalamus (T) they ascend to the cortex (*top*) where there are areas of representation as yet undetermined or delineated.

Theories about the physiologic system postulate that all messages are transmitted within the nervous system as electrical impulses<sup>14</sup> and act across numerous synapses (Fig. 10-7). These messages are termed *neurotransmitters*. The postsynaptic neuron depolarizes thousands of signals from the synapse and generates an action potential that proceeds along the neuron. This was the neuron concept of Waldeyer and Cajal.<sup>15</sup> Golgi rejected this concept and proposed the concept of a “functional syncytium.”<sup>16</sup> Besides transmission through a neural network (Fig. 10-8) it is also postulated that electrochemical signals are transmitted via extracellular medium (Fig. 10-9). The wiring system concept fails to support morphological coupling between neurotransmission release sites and receptor sites (Fig. 10-10).<sup>17,18</sup>

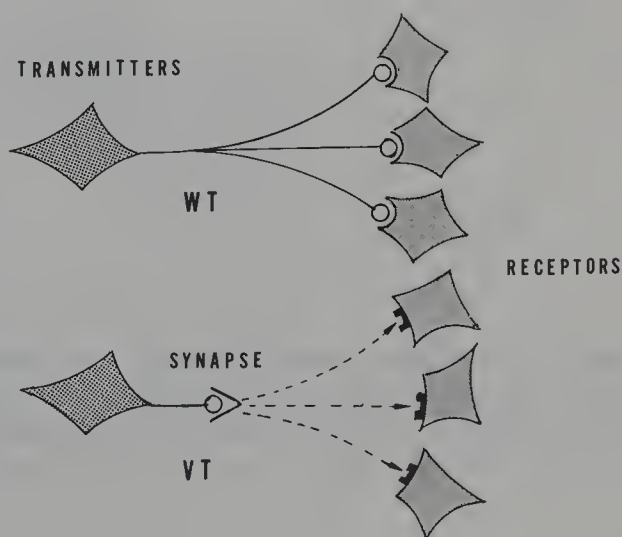
Figure 10–7. The synapse. In wiring transmission (WT) the fiber terminates in a preganglionic bulb (PR) that contains numerous vesicles (V) containing acetylcholine. When this choline is liberated it passes across the synaptic space (SS) causing a depolarization of the postsynaptic membrane (PS), resulting in further transmission of WT impulses. The choline within the synaptic space initiates an impulse transmission termed volume transmission (VT).



The wiring transmission concept requires the existence of specific receptors to receive specific impulses, indicating the theory that a code must exist.<sup>19</sup> The volume transmission system appears more probable, although the impulses are transmitted at a lower speed. The energy needed for this transmission has not been confirmed.

Stress has been invoked as objectively and subjectively influencing pain perception. There are two neuroendocrine systems involved in this mechanism: the hypothalamo-pituitary-adrenocortical and the hypothalamo-sympatho-adrenocortical systems via a corticotrophin-releasing hormone (CRH).<sup>20,21</sup> Both the volume and the wiring transmission systems are involved in this transmission. This hormonal involvement is the basis of Chapter 8.

Figure 10–8. Transmission of wiring transmission compared with volume transmission. Wiring transmission (WT) indicates specific connections made from transmitter to specific receptor organs. Volume transmission (VT) indicates chemical neurotransmission leaving the synapse and finding receptors wherever they are or as numerous as they may be. VT does not replace WT but merely enhances the other.





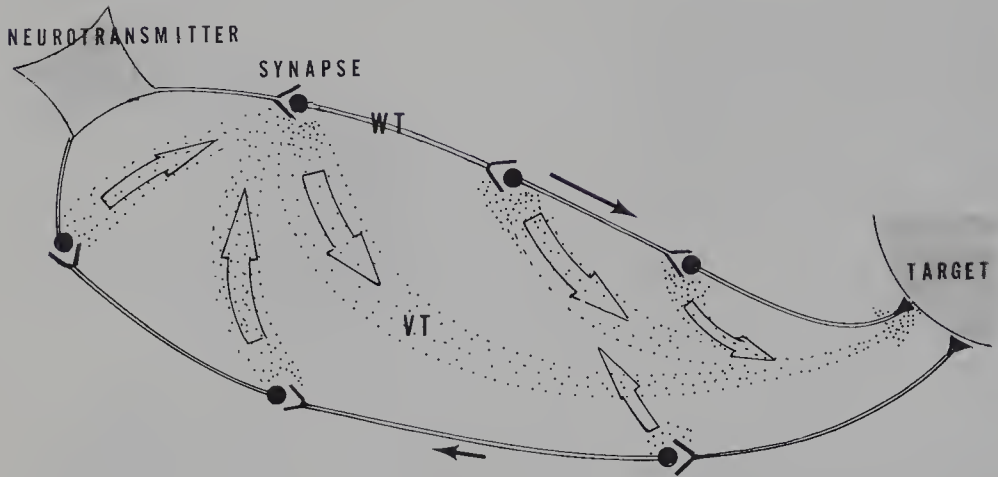


Figure 10-9. Schematic wiring transmission compared with volume transmission. Direct neurotransmission by wiring concept (WT) shows direct connection to the receptor organ (*dark arrows*). At each synapse chemical neurotransmitters are released that enter the interstitial space and chemically locate their receptor organs (*white arrows*). Volume transmission (VT) goes in both or either afferent or efferent direction.

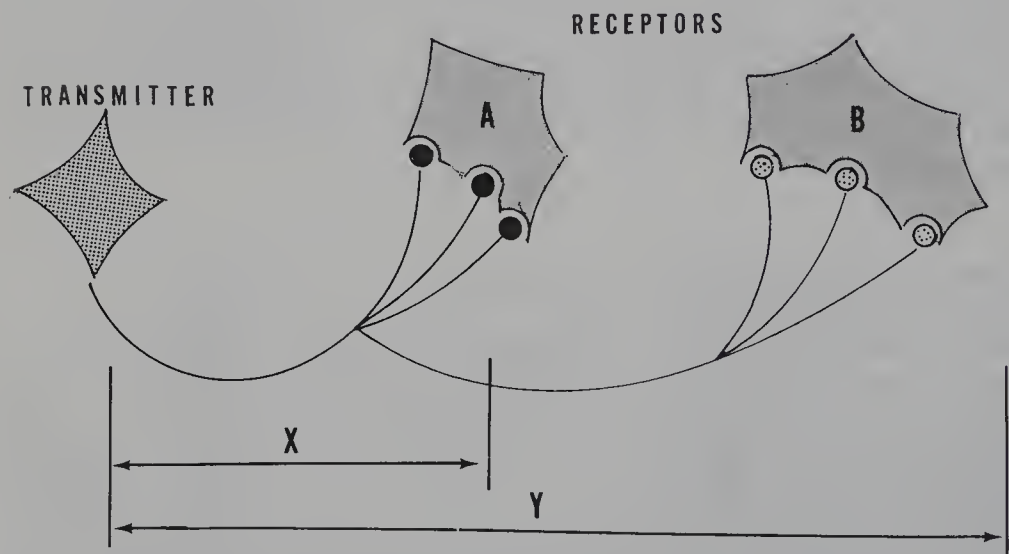


Figure 10-10. Mismatched transmitter and receptors (concept). Mismatching between neurotransmitter and receptors may occur if the impulse goes distance X to activate specific receptor A and proceeds distance Y to later activate receptor B. Each receptor may vary in its reaction potential.

In understanding acute pain before dwelling on chronic pain, there are other aspects that need clarification. The concepts of pools within the nervous systems has been advanced (Fig. 10–11). These pools also indicate the sites of action on the treatment of pain (Fig. 10–12).

The reflex muscular spasm that accompanies local pain also can become a site of nociception (Fig. 10–13) that aggravates the pain and leads to disability (Fig. 10–14).

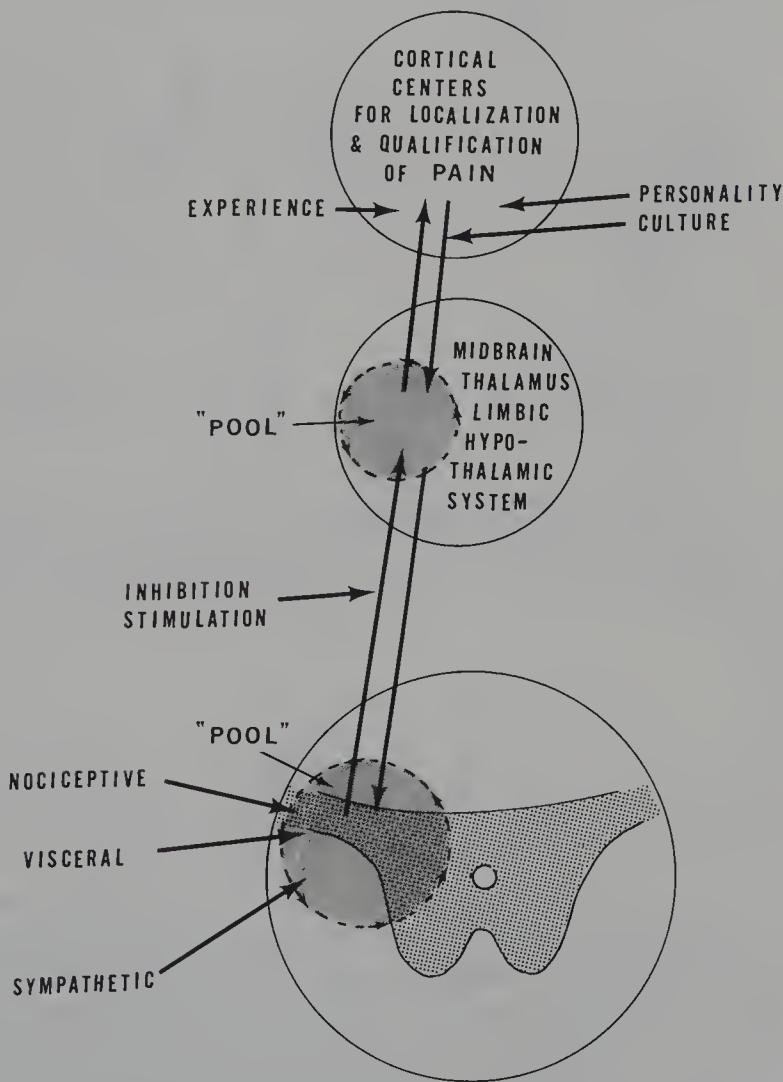


Figure 10–11. Sensory center pools in the modulation of pain. Pool centers are postulated in the modulation of pain, beginning at the cord level and ascending to the thalamic area and finally the cortex. Various aspects of the perception of pain are indicated.

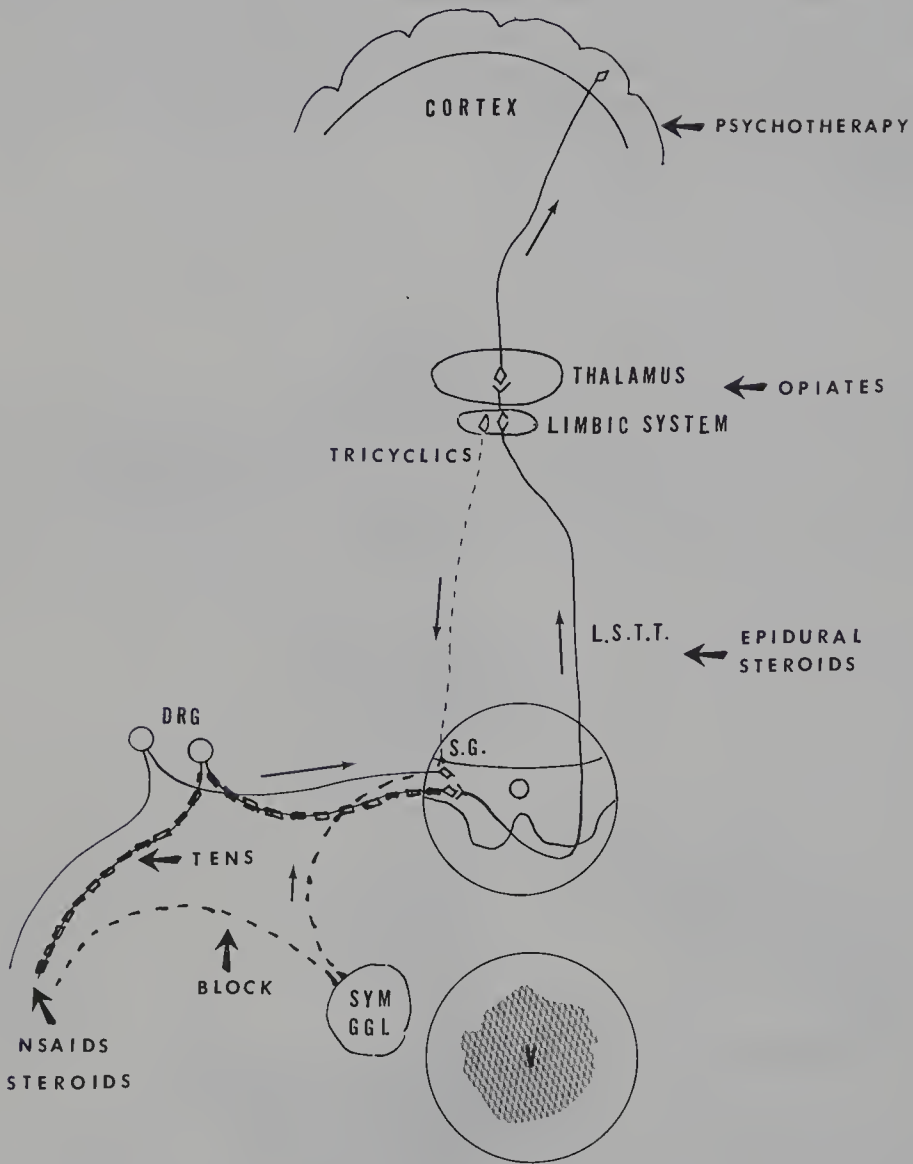


Figure 10-12. Sites of action in the treatment of pain. Anti-inflammatory agents (steroids and NSAIDS) act at the tissue site of injury. TENS acts at the peripheral area from the tissue site and the dorsal root ganglion (DRG) prior to entry at the cord level (SG). A sympathetic nerve block (block) interferes with sympathetic impulses originating at the sympathetic ganglia (GGL). Epidural steroids act at the cord level of ascending lateral spinothalamic tracts (LSTT). Tricyclic medication acts on the thalamic, hypothalamic (limbic system) level whereas psychotherapy intervenes at the level of the cerebral cortex.

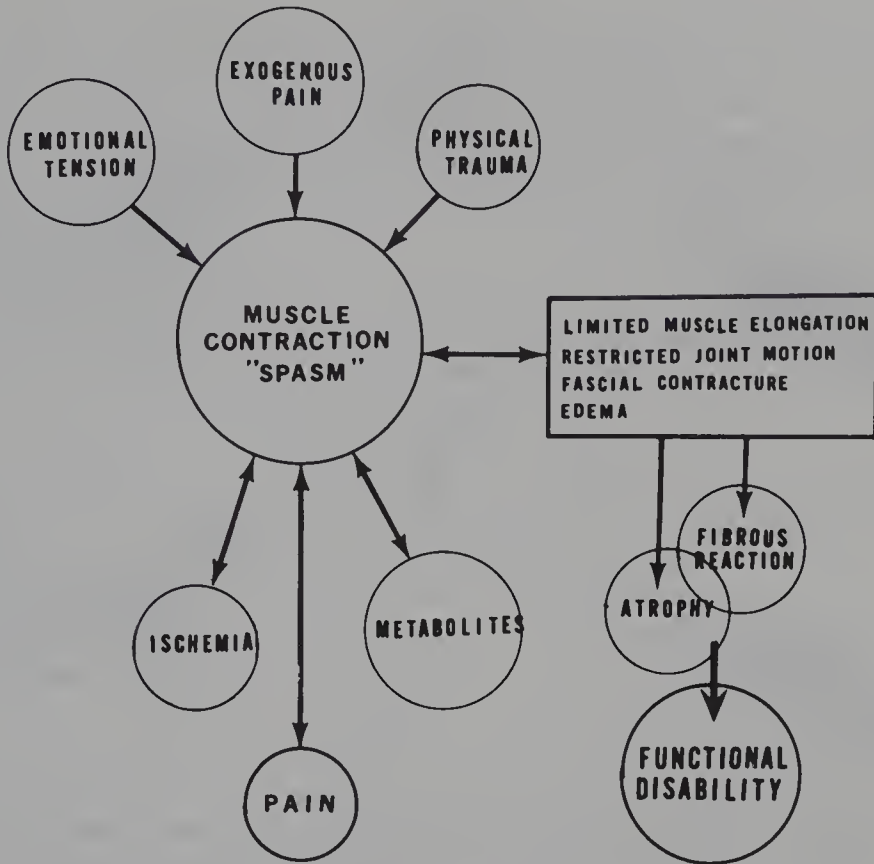


Figure 10-13. Sequence of changes from sustained muscular contraction (spasm). Muscular contraction (spasm), initiated by tissue damage and resultant pain, causes sequences of tissue reactions that lead to functional disability.

The final pathway is obviously the brain, where the pain is interpreted. Clinical studies on how and where in the brain pain is interpreted are being done. The research goals are (1) to find meaningful qualitative and quantitative data on where the brain changes when pain persists, (2) to determine how these markers can be used to monitor pathogenesis and effects of treatment measures and also afford meaningful prognostic data, (3) to cast light on neurophysiological and neurochemical mechanisms that underlie pain concepts, and (4) to afford noninvasive measures to determine the relationship of human to animal pain experiences.<sup>22</sup> These are *pain correlates*, which mean markers or concomitants of neurophysiological activities associated with pain experiences.

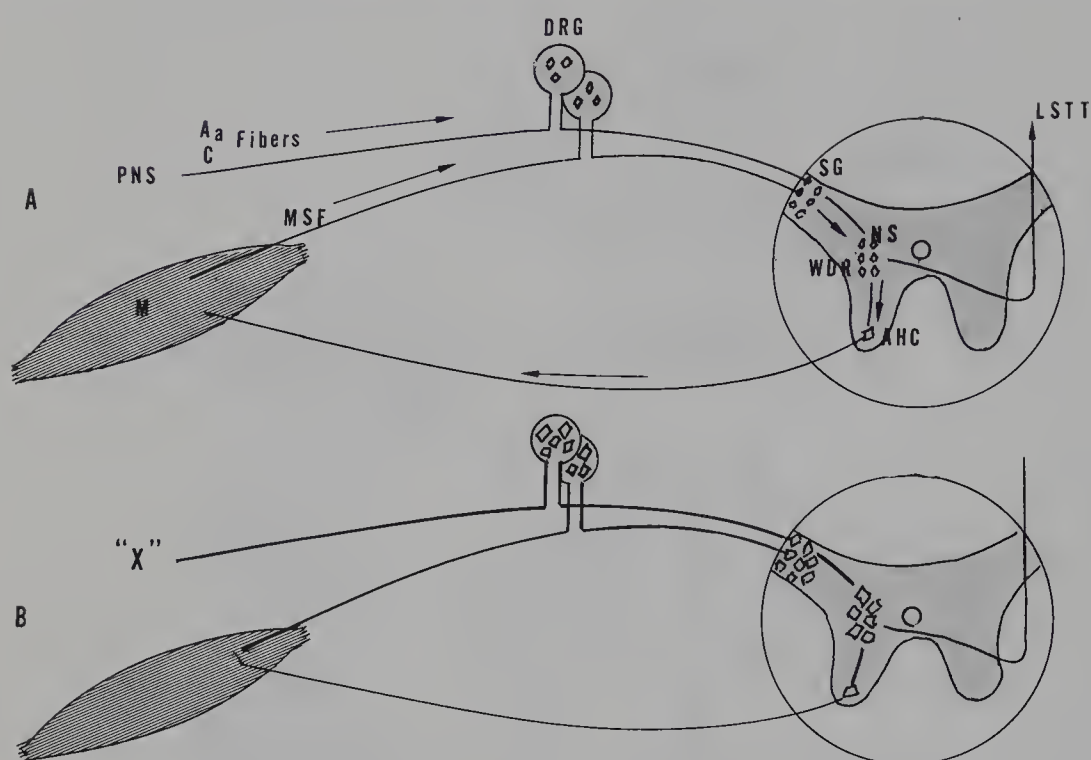


Figure 10-14. Expanded receptive fields of hyperalgesia. *Panel A* depicts the accepted routes of nociception from peripheral nociceptive sites (PNS) ascending via C and A alpha fibers through the dorsal root ganglion (DRG) into the substantia gelatinosa (SG). From this site there is transmission to nonspecific neurons (NS) with reaction of wide dynamic ranges neurons (WDR). Interneuronal pathways then excite the anterior horn cells (AHC), which cause muscular contraction (M). Sensory impulses from the muscles then ascend via muscle afferent fibers (MSF) to complete the sensory cycle of pain. *Panel B* depicts that repeated impulses from the periphery X increases the size and number of neurons within all these sites (the DRG, NS, and WDR) with increased sensitivity of the anterior horn cells, which are greater, and prolonged muscular contraction.

Biological measures have included topographic mappings and tomographic imaging, which thus far have failed to ascertain validity and reliability to neurophysiological correlates.<sup>23</sup> At this stage in research they include brain evoked potential (BEP), magnetic evoked fields (MEF), cortical power spectrum (CPS), renal blood flow (RBF), computed axial tomography (CAT), magnetic resonance imaging (MRI), positron emission tomography (PET), single-photon emission computed tomography (SPECT), and nuclear magnetic resonance spectroscopy (NMR). To date there is no brain measure that correlates neurophysiological activity to clinical pain, but there is promise.<sup>23</sup>



Chronic pain was originally considered to be pain that lasted longer than 3 months, but chronic pain is now considered to exist in significantly shorter periods of time. As chronic pain occurs as a prolongation of acute pain, all the mechanisms so far discussed must be addressed to prevent pain from becoming chronic.<sup>24</sup> Proper treatment of acute pain can prevent chronic pain before it becomes deeply engrammed in the central nervous system.

In a classic dissertation, *The Culture of Pain*, Morris<sup>25</sup> states that pain pervades all aspects of human social life and emotions and has done so since antiquity. Aristotle wrote in *De Anima* that “pain upsets and destroys the nature of the person who feels it.”

Acute pain must be addressed in all its aspects, otherwise chronicity and disability will prevail (Fig. 10–15). All interventions to minimize the chemical, mechanical, and electrical effects of the algogens on the neurophysiological conduction of nociception must be applied. This must be done with the appreciation of the psychosocial implications of the pain from the very onset. Treating the symptoms on the basis of a purely mechanical chemical basis from impaired neuromusculoskeletal mechanism is fraught with ultimate failure. The patient must be treated, not merely the symptoms, the clinical findings, or the diagnostic tests. The acute symptoms must be addressed, but always with the need to instruct the patient of the meaning of the symptoms, the significance of the resultant disability, and the role to be played by him or her as the procedure ensues.

The noxious agents that accumulate at the peripheral trauma site, comprising histamines, kinins, neuropeptides, and numerous other algogens, have a vasomotor effect: either vasodilation or vasoconstriction.

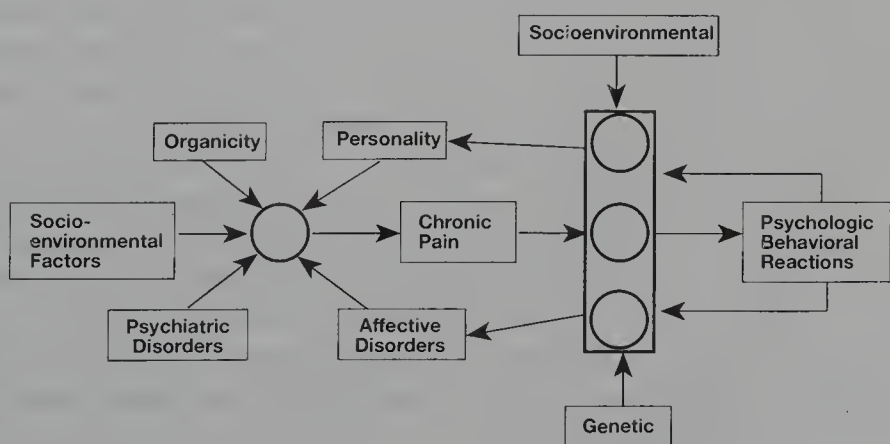


Figure 10–15. Concept of chronic pain. Factors contributing to chronic pain.

These nociceptive agents lower the threshold of the receptors of the A delta and C afferent fibers of the peripheral nervous system, sending impulses cephalad. Intervention or elimination of these algogens at the trauma tissue site is the objective of the application of most modalities.<sup>26</sup>

The local area becomes hypersensitive, creating pain known as primary hyperalgesia. A secondary hyperalgesia occurs in the surrounding tissues from antidromic activation of the C primary afferents, which release substance P in the region.<sup>27</sup> This hypersensitivity may also be an afterdischarge from small primary afferents that release substance P. Electrical stimulation of the primary afferents has been shown to release substance P at their receptor ends. Reactive local skeletal muscle spasm occurs, initiating a pain-spasm-pain cycle.

The chemical and mechanical substances produced at the peripheral tissue site following injury are mechanisms of pain production that must be addressed in the treatment of acute pain.

## PHYSICAL MODALITIES

### Cryotherapy

Cryotherapy, the application of cold in the treatment of acute treatment of pain, has been an accepted modality for centuries. Three possible mechanisms for the effectiveness of local cold have been (1) receptor adaptation,<sup>28</sup> (2) counterirritant effect,<sup>29,30</sup> and (3) a neurogenic effect.<sup>31,32</sup> To date, there is no evidence that cold thermal agents activate the endorphin system, which has been postulated.

Cold lowers the temperature of the skin and underlying tissues essentially by removing heat from these tissues. Theories behind its local application as a treatment modality for a traumatized area are that (1) it decreases or inhibits bleeding; (2) it decreases local tissue metabolism, which produces algogens; (3) it neutralizes the local histamine liberated by trauma; (4) it decreases local muscle spasm by decreasing the sensitivity of the muscle spindle system<sup>33</sup>; and (5) it elevates the threshold of pain-transmitting nerves.

A local tissue reaction to trauma is the formation of local edema. Edema occurs because of change of hydrodynamics as the vasoconstriction is followed by reflex vasodilation. The afflicted vessels, the arterioles, the capillaries, the venules, and the lymphatics become distended. The endothelial cells separate, creating gaps between the cells and allowing greater filtration of the serum with its contained constituents to enter the perivascular tissues.

This edematous fluid at first is merely a transudate containing water and dissolved electrolytes. It has a specific gravity of 1.012, maintaining osmotic balance. As permeability increases, the transudate becomes an exudate, containing cells and protein, with a specific gravity of more than 1.012, which causes an imbalance of osmotic pressure, causing further flow outward into the perivascular tissues.

Both the transudate and the exudate cause a mechanical impedance to further blood flow, resulting in ischemia. The protein contained in the exudate gradually causes chemical “thickening” of the fluid, which impairs physiological movement between fascial planes.

Ice or cold applied to the inflamed tissues intervenes in this transudate-exudate cycle by decreasing fluid transudate and decreasing metabolic rate. Cold also decreases tissue sensitivity, which permits active and passive exercises that mechanically express the exudate and transudate from the tissues.

Nerves differ in their reaction to cold,<sup>30</sup> depending on their degree of myelination. The unmyelinated, small-diameter fibers are less responsive to cold than are A fibers, with the large motor fibers (alpha) the least affected.<sup>31,32</sup>

Exercise performed after the application of cold generates more muscular tension.<sup>33</sup> The combined effect of ice, therefore, decreases pain, alters the hydrodynamics, and permits greater strength of muscle contraction, decreasing the accumulation of edema and removing the accumulation of the nociceptive metabolites.

## Therapeutic Heat

The sequelae of the effect of heat on tissues depends on the extent of temperature rise, the rate of application of heat energy and the volume of tissue exposed to the heat application.<sup>34</sup> Elevation between 40°C and 50°C increases blood flow, which is the therapeutic objective.

Increased blood flow causes warmer blood to reach the site and remove the cool blood. The rate of temperature rise influences its efficacy. A slow rise of tissue temperature may defeat the objective of heat application, as it brings cooler blood into the inflamed tissue site. Too rapid a temperature rise may also be deleterious, as the heat generated in the local tissues may stimulate pain receptors with adverse effect.

The effects of heat can be stated to be an alteration in metabolic activity, hemodynamic function, neural response, skeletal muscle activity, and modification of collagen tissue.<sup>35</sup> All these effects can be di-

rectly or indirectly related to the management of pain resulting from tissue trauma. The neural response more directly intervenes in transmission but the other effects of heat relate to the tissue dysfunction, which also enhances pain.

The neural effect of how heat provides analgesia and reduces muscle spasm, both involved in pain production, is not fully understood.<sup>36,37</sup> The latter, reduction of muscle spasm, is conceivably induced via the spindle system.<sup>38</sup> Heating the area over a peripheral nerve by high-intensity infrared radiation has induced analgesia distal to the application.<sup>39</sup> Much research remains to be performed relative to the precise neurophysiological basis for relief from pain, but clinical experience seems to claim the effectiveness of heat.

Surface heating agents do not elevate muscle temperature needed to alter II or Ib afferent nerve activity, whereas skin temperature heating has decreased gamma efferent activity,<sup>39</sup> which may relate to diminished muscle spasm related to pain reduction.

Metabolic rate increases two- to threefold with every 10°C rise. To increase the tissue temperature above 50°C burns the tissues, as their repair potential cannot cope with the protein denaturation of excessive heat. Chemical and metabolic activities are beneficially increased below that temperature.

The hemodynamic effect, increasing blood flow, occurs as superficial heat causes a reflex postganglionic sympathetic nerve activity to the smooth muscles of the blood vessels, supplying more blood flow to deeper organs such as muscle.<sup>40</sup>

The most effective heat modality proposed for therapeutic intervention has comprised voluminous medical literature<sup>41</sup> and will not be thoroughly evaluated here.

Moist heat transmitted via hot moist packs has many advocates.<sup>42</sup> Local hot paraffin wax is effective in treating extremities, as are hydrotherapy<sup>43</sup> (but not used in the low back), ultrasound,<sup>42</sup> diathermy, pulsed electromagnetic fields, and laser. Whimsically, Licht<sup>44</sup> commented that "the choice of source of heat will depend on the training and experience of the physician, or empirically: the latter, a matter of local routine often based, regrettably on such considerations as cost, availability, convenience, hand-me-down habits, custom or publicity."

## Soft Tissue Modalities for Elongation

Connective tissue, which is so often impaired after injury or disease, is benefited by heat application. Connective tissue, be it collagen, elastin, or fibrous tissue, tends to shorten after injury.<sup>45</sup> The viscoelastic property of connective tissue that permits elongation from physical



stretch is known as *plastic deformation*.<sup>46</sup> Recoverable deformation is possible if the modalities of heat and passive-active stretch are applied to the deformed (shortened) tissues.<sup>47</sup>

The need to regain tissue flexibility in treating pain is apparent in that sensory nerves are enclosed within the soft tissues, which often have become impaired after injury or prolonged tension from anxiety, anger, and emotional tension.

To regain the physiological elongation of damaged tissues to their normal length requires ensuring the appropriate temperature elevation as regards intensity, site, and duration. Consideration must also be given as to the extent of physical stretch regarding its intensity, duration, and velocity.<sup>48</sup> The techniques of stretch have been propounded, varying from (1) constant load to overcome impaired elasticity, to (2) rapid stretch followed by holding the gained elongation, to (3) a slow progressive stretch.

## Analgesic Nerve Block in Treating Pain

The original text of Bonica's *The Management of Pain* stated in its title "With Special Emphasis on the Use of Analgesic Block in Diagnosis, Prognosis and Therapy."<sup>49</sup> This laid the groundwork for interrupting all peripheral tissue sites of nociception by analgesic nerve blocks. This concept is still valid to acute and recurrent pain for its diagnostic localization, treatment, and even prognosis.

Diagnostic blocks, albeit less sensitive and accurate than diagnostic imaging and electrodiagnosis,<sup>50</sup> is valuable in conforming structural abnormality. If the structure or tissue is ascertained, interruption of its nerve supply can be therapeutic as well as diagnostic.

The purpose of analgesic nerve blocks is to interrupt the transmission of nociceptive impulses of the afferents to the cord from the damaged tissues. Diagnostically the afflicted organ or tissue is identified by interruption of the somatic nerve to and from that tissue. Acute pain is interrupted and therapeutic procedures on that organ or tissue are permitted during the period of analgesia-anesthesia.

Interruption of the afferent impulses in the treatment of acute pain allows a more normal healing process or, at least, allows comfort to that patient during the healing process.

Because repeated or continuous impulses from the end organs (terminals) increase the sensitivity of those fibers, the dorsal root ganglia, the dorsal column neurons, and even the thalamic pathways, interruption of the initial barrage of impulses diminishes the subsequent sensitivity enhancement.

In the treatment of acute low back pain, with or without leg radia-



tion, the site of nerve block presents a clinical problem. Bonica<sup>49</sup> in his initial text states that “analgesic blocking, skillfully performed, can play a significant role in the management of acute low back pain, regardless of the etiology.” He proceeds with the statement, “it is . . . an important adjunct to other treatment . . . even though it may produce complete and permanent relief of pain.” He stresses that analgesic blocks are effective in relieving muscles spasm “as known.”

The analgesic blocks (Bonica) are administered into the tender muscle(s) and into the ligaments of the apophysial, sacroiliac, and sacrococcygeal joints (Fig. 10–16), which do not interrupt the nerve supply to the affected tissues.

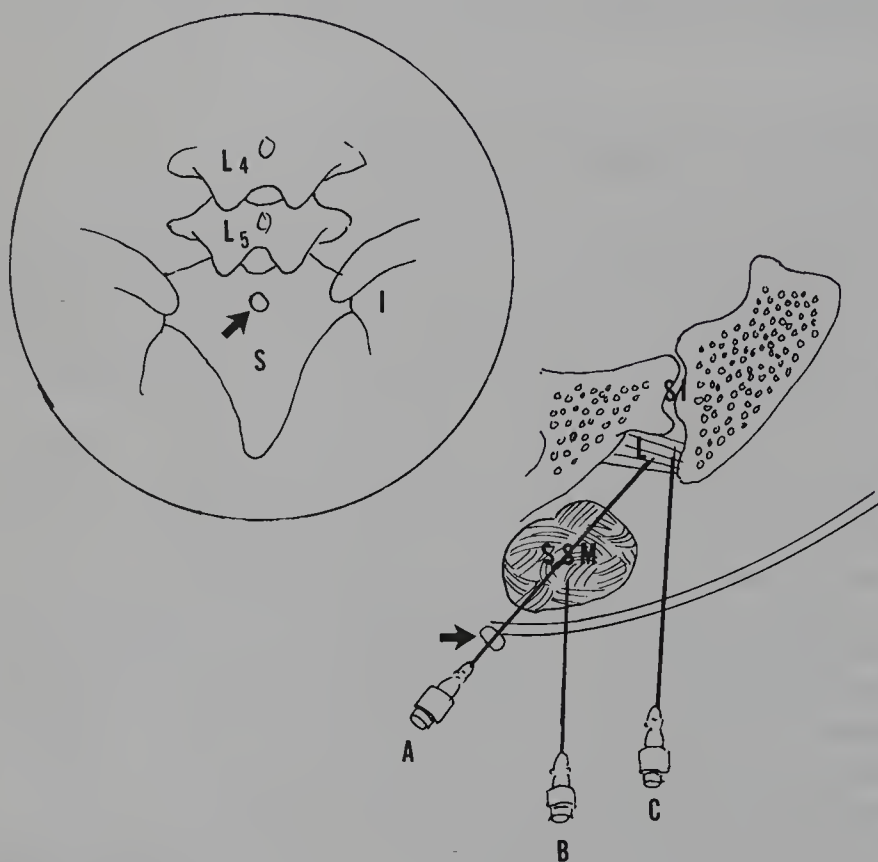


Figure 10–16. Sites of injection. Analgesic and steroidal injections into nociceptive tissues of the lumbosacral area are shown. *A* penetrates the sacral muscles (SM) to invade the iliosacral ligament (L). *B* penetrates the muscles and *C* infiltrates the ligament (L) and goes deeper into the sacroiliac joint (SI). In the encircled view *S* is the sacrum, *I* is the ilium, *L5* is the fifth lumbar vertebra, and *L4* is the fourth lumbar vertebra. The dot (arrow) is the site of entry for an epidural injection.

Of neural blocks, the caudal and peridural blocks are effective but are usually considered only when pain persists and does not respond to other modalities. Perivertebral blocks also fall into this category.

The techniques of performing nerve blocks have been extensively described in textbooks of anesthesia and acute pain.<sup>49</sup> The precise nerve supply of every tissue in the lumbosacral spine is well known, but technically interrupting this nerve supply is not in the domain of the average clinician, nor is it necessarily indicated as an initial treatment procedure.

It is well documented that the analgesic value that occurs during the presence of the injected drug continues after the chemical duration of the drug. The basis for this prolongation remains obscure.

A recent controversy has been raised as to the psychophysiological benefits involved in nerve blocking for chronic pain.<sup>51</sup> This may be an oxymoron, as any modality that is beneficially used in treating pain remains used regardless of its mechanism.<sup>52</sup> The numerous medications injected are well documented in the literature, but even this raises questions, as there is benefit from insertion of a needle without the use of medication. The "dry needle" has proved effective.<sup>53</sup>

The needle used here may be a placebo or effective as to its neurophysiological aspect. The pain relief obtained must be evaluated carefully to avoid ascertaining that the nerve blockade of the pain was objective and conclusive. "The placebo effect . . . is most powerful when a trusted physician enthusiastically offers a patient a new therapy."<sup>54</sup>

Injection tests have been employed to determine patient response before considering indwelling epidural injections and/or surgical procedures for chronic pain.<sup>50</sup> Once the catheter is inserted, an injection of normal saline is introduced. If there is relief of pain, further blocks are not undertaken. Further injections of lidocaine hydrochloride of varying strengths are then introduced. This is essentially a psychological determination preceding a more definitive procedure to ensure its success, and its value is questioned by many.

Discogram, injection into the nucleus of a disk, has been advocated as a diagnostic test. In spite of the fact that injecting a normal disk can create significant pain, this procedure has continued to be advocated. It has value in the production of "the pain suffered by the patient" and in localizing a specific disk level.<sup>55</sup>

It is postulated that certain situations justify discography<sup>50</sup>:

1. Discography may be justified in the case of a severely disabled patient who is being considered for surgery with multiple-level disk disease demonstrated by scans. Prior to surgery, a disco-

- gram can help define the multilevel disk abnormalities that are painful and that most exactly reproduce the patient's usual pain.
2. Prior to surgery, a discogram can verify that a lumbar-level adjacent to a proposed fusion is normal and can support a fusion.
  3. In the rare case of internal disruption, or another discogenic syndrome that has strong consistent history and failure of conservative care, the scanning procedure will be relatively normal as will be myelography, bone scan, and electromyogram (EMG). It is a good idea to have psychological studies performed.

These three postulates are vague and assume that the contemplated surgery is indicated and can even be considered as appropriate: "In summary, surgery for lumbar spine pain or low-back pain with radiating leg pain has a notoriously unsuccessful track record. . . . Studies have shown that time alone may cure herniated disks for which surgery has been indicated."<sup>56,57</sup>

It can be summarized from extensive review of the literature that injections are limited to diagnostic procedures, facet etiologies, discograms, and epidural analgesia.<sup>58</sup>

The technique of epidural injection is well documented<sup>59</sup> (Fig. 10-17). The benefit is limited, and its value is subjectively palliative albeit of temporary value.

Epidural corticosteroid injection has proved to be a relatively effective treatment for low back pain (and sciatica). There has been a low incidence of side effects, but its outcome assessment remains unproved.

The Waddell nonorganic signs (WNOS) must always be considered in determining the outcome. These are skin and subcutaneous tenderness, pain simulation from axial loading and axial rotation, straight leg raising (without dural signs), lumbar movement limitation and motor weakness (with and without distraction), sensory abnormalities, and overreaction.<sup>60</sup>

Pain relief lasting 3 weeks and a likelihood of return to work in 3 months has been documented.<sup>61</sup> The procedure is usually well tolerated and the incidence of arachnoiditis is extremely rare. The patient with low back pain is benefited, but for those with sciatica it is less effective.

## Exercise in Treatment of Low Back Pain

For many years exercise has been the major conservative modality in the treatment of low back pain being advocated for acute pain, for the prevention of recurrence, and even for the management of chronic low back pain.

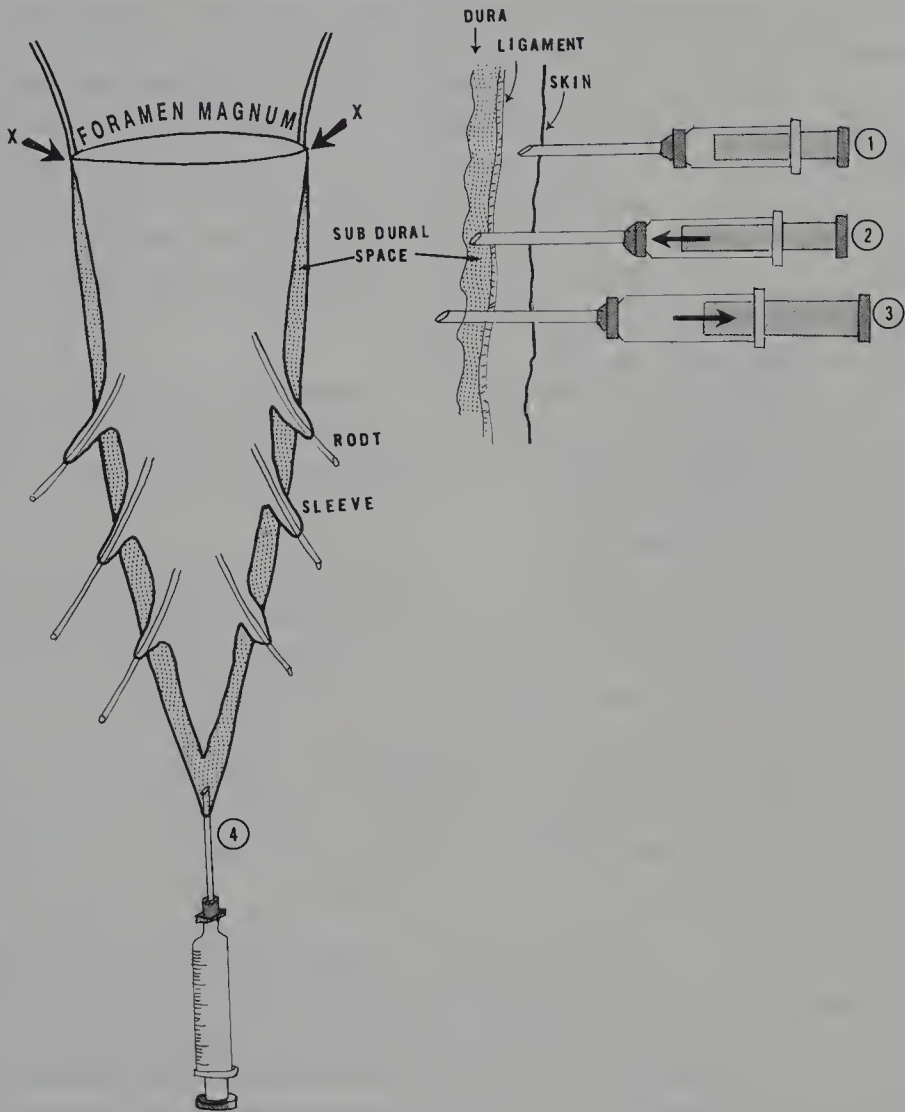


Figure 10–17. Epidural injection. The dural sac is closed at the foramen magnum (X) then descends distally, enclosing the nerve roots as they emerge through the foramina. The technique of injection: (1) spinal needle penetrates the skin into the subcutaneous tissues towards the dura, at this stage there is no pressure change in the syringe; (2) penetration through the ligament into the subdural space where pressure is less and the plunger enters the syringe; (3) if the needle penetrates the dura it becomes an intradural (spinal) tap and the spinal fluid pressure is higher and forces the plunger out of the syringe; (4) another epidural route is at the caudal tip through the sacral notch.



There persists a controversy as to which exercise is appropriate in treating the various back pain syndromes. There are advocates of flexion exercises, extension exercises, aerobic exercises, stretching regimens, diminution of tension, and exercise as an aspect of normal low back function. The controversy is augmented in respect to active versus passive therapy.<sup>62</sup> Outcomes assessment of exercise regimens, have been undertaken recently, with many articles appearing in medical journals.<sup>63-71</sup>

In an evaluation of the physical management of low back pain,<sup>72</sup> many modalities are discussed, but exercise is not mentioned. In a provocative article, Tollison and Kriegel<sup>73</sup> posed two questions: (1) what evidence is there to support the claim of the value of exercise, and (2) which exercises have proved beneficial?

These questions were raised in relationship to the Williams exercises,<sup>74</sup> then the classic exercise treatment protocol. The author<sup>75</sup> discussed exercises in the context of "prevention or recurrence." Cady<sup>76</sup> firmly established physical exercise and general fitness in the prevention of back injuries and pain.

The literature on exercises in relationship to low back pain has had the following objectives: (1) to decrease the duration of the impairment and thus disability, (2) to strengthen and increase endurance, (3) to reduce mechanical stress (ergonomics), (4) to correct posture, (5) to bring about general restoration, and (6) to reduce pain.

For reduction of duration of pain and impairment, the exercise prescribed in the traditional model is "exercise according to pain severity . . . let pain be your guide." Fordyce et al.<sup>77</sup> initiated exercise at a fixed time not at a fixed symptom, with a significant decrease in duration of disability.

Increasing strength and endurance of back muscles have become the cornerstones of treatment of low back pain<sup>78</sup> (see Chapter 7) with mixed outcome assessment. The duration of back pain appears to correlate with the resultant muscle strength and endurance.<sup>79</sup> There was an insignificant difference in strength between normals and patients with symptoms for less than a month.<sup>80,81</sup>

DeVries,<sup>82</sup> doing EMG studies, concluded that muscular deficiency could be a causal factor in low back pain and that muscular fatigue could also be significant.<sup>83</sup>

All these studies on back muscle strength and endurance indicate that proneness to back injuries is influenced by deficiency but fail to indicate the value of exercise once the patient becomes symptomatic.

Reduction of mechanical stress (ergonomics) with exercise also remains controversial. "Body mechanics" involve eccentric muscle contraction—elongation and deceleration (see Chapter 1). Strengthening exercise directed at the abdominal (flexors) allegedly decreases the load

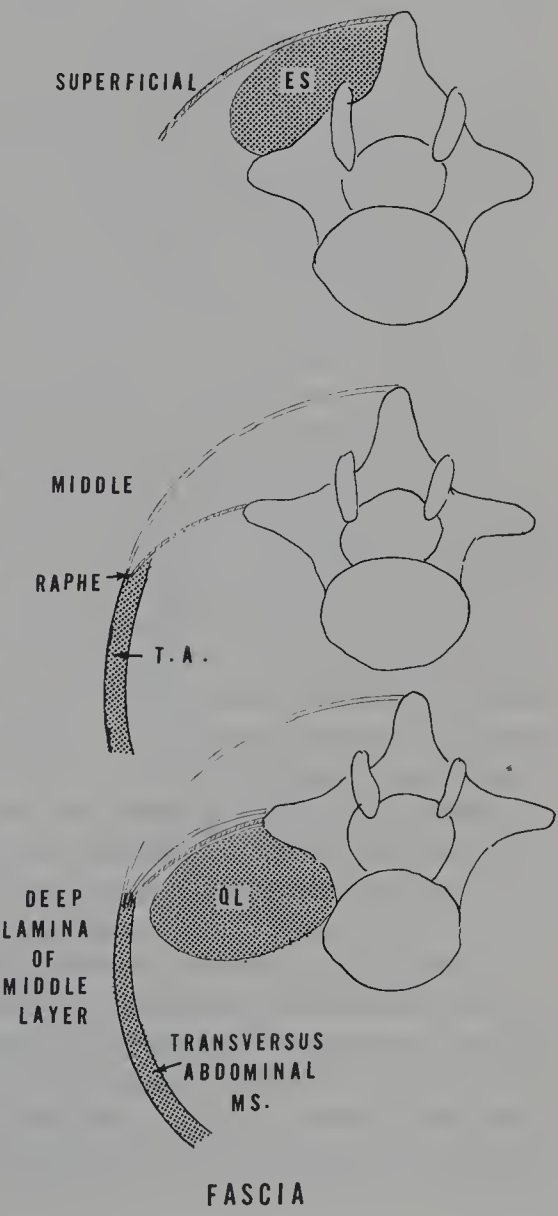


upon the disks,<sup>84</sup> but how this effect occurs in isotonic flexion exercise remains unconfirmed.<sup>85</sup>

A concept of the value of the oblique abdominal muscles<sup>74</sup> is the effect of the oblique muscles upon the fascia (Fig. 10-18). This concept implies that the value of the exercise is not during the lifting but as a strengthener of the tissues used during a lifting episode.

Poor posture, when it is considered a cause of low back pain, supposedly is improved by exercise, but this remains unconfirmed.<sup>86</sup>

Figure 10-18. Fascial component of intracompartmental force on the low back. The fascia of the low back muscles that function kinetically also contribute to pain when inflamed or causing compartmental pressure. The fascia is divided into superficial layers, containing the erector spinae muscles (ES). A raphe exists that divides the fascial of the transverse abdominal muscles (TA) into a middle sheath. The deep lamina of the middle fascia covers the quadratus lumborum muscle (QL). Each of the fascial lamina form compartments.



Pain reduction in the low back remains predominant in treatment protocols. The statement that "exercise therapy is the cornerstone treatment for subacute and chronic pain"<sup>87</sup> is quoted in Bonica's classic volumes on the management of pain.<sup>88</sup> The statement continues: "During acute pain exercise generally is contraindicated except for maintaining selfadministered passive range of motion (ROM) of all extremities and the trunk." Subacute pain, however, "is less intense . . . therefore therapeutic exercise is highly desirable and realistic 'for restoration of function to the affected area.' " This statement implies that exercise is mainly directed to the functional impairment of trauma and/or illness and only indirectly applies to the relief from pain.

In the treatment of chronic pain, exercise is directed to the effects of decreased activity, leading to atrophy, weakness, contracted joints, and so on, with pain only indirectly addressed. In Basmajian's<sup>89</sup> treatise on *Therapeutic Exercise* the use of exercise in treating painful syndromes is directed at specific organ systems without mention of exercise as a modality treating pain per se. DeVries<sup>90</sup> in his discussion of the physiology of exercise does not mention pain.

Fordyce,<sup>91</sup> whose operant conditioning has become an accepted effective treatment procedure in the treatment of chronic pain, uses exercise only to increase activity level. He states that "exercise is, with few exceptions, also a behavior that is incompatible with pain behavior."

There is no doubt that muscular weakness, prevalent in many musculoskeletal pain syndromes<sup>92</sup> as well as fatigue and debility that occur in depression from chronic pain, can be altered, in part, by exercise. In that respect, exercise to regain strength and endurance as well as flexibility and mobility is a powerful adjunct in the treatment of pain.

It has been implied that exercise increases the level of endorphins (endogenous opioid peptides),<sup>93</sup> which are accepted neurotransmitters with a morphinelike action.<sup>94</sup> This modulation of pain perception and analgesia has been associated with the analgesia of electrical brain stimulation and acupuncture.<sup>95</sup>

Exercise results in elevation of adrenocorticotrophic hormone, cortisol, and catecholamines, which are the precursor of beta-endorphins. Many athletes have had a decreased pain perception that has allowed them to make maximum effort in spite of pain and even claimed to reach an emotional high after extreme physical activity.<sup>96</sup>

Naloxone has altered pain perception experienced after running.<sup>97</sup> Naloxone, a narcotic antagonist that competes for endorphin-binding sites, substantiates that endorphins are acting in the athlete. After 30 minutes of strenuous exercise, there has been noted an elevation of plasma endorphins. Further studies have refuted these assertions when

they found that mood changes attributed to endorphins after a 10-mile run were evident with and without naloxone.<sup>98</sup>

Pitts,<sup>99</sup> studying the effects of emotions and thus implying the association of endorphins in stressful exercises, postulated that the accumulated lactate from maximal exercising induces anxiety, which has a mood-induced endorphin effect. This again has been refuted in the finding that elevated endorphins after stressful exercise may be noted without significant elevation of lactates.<sup>98</sup>

In a recent paper, Padawer and Levine<sup>100</sup> revealed that in their experiment they found no analgesic effect from exercise but that the analgesic effect was from the pain "pre-testing" itself. In this experiment their subjects had been exposed to pain pretesting; those tested exhibited analgesia from the test alone. Exercise following did not alter the analgesia.<sup>101</sup>

The conclusion of endorphin relationship with mood changes and analgesia related to strenuous exercise is not yet ascertained, but there is value in further studies. What, so far, appears apparent is that exercise must be strenuous to be analgesic, and, therefore, what its role will be in treatment of pain will depend on the physical and psychological ability of the afflicted patient to implement this modality when confronted with persistent or chronic pain.

Exercise has a well-documented place in the physical treatment of most neuromusculoskeletal painful disabling conditions, but its precise role in the treatment of acute low back pain remains unclear. Its role in prevention of recurrent low back pain is more acceptable but as yet unconfirmed in outcomes assessment as to type and frequency.

This should not be considered as refuting exercise in treating patients with low back pain but rather a plea for careful scientific outcomes assessment in evaluating patients with this complaint. The fact that exercise is administered with a personal regard for the patient and with information and instruction to the patient undoubtedly plays a large role in either benefit or lack of benefit to the treatment program.

Discipline of the patient performing the prescribed exercise also needs evaluation. Exercise requires effort and dedication and a promise of value for the effort. The psychological effect is evident. In today's economic evaluation of any prescribed medical care, it behooves professionals to carefully evaluate their prescribed modalities.

Evaluation of treatment protocols on the natural history of low back pain and return to work has failed to demonstrate any specific beneficial results.<sup>102-104</sup> It seems that if pain persists for more than three months the psychological makeup of the patient changes.<sup>105,106</sup> Relief of pain and improvement in function must be initiated and ensured early in the process to prevent debility. Exercise alone does not appear very effec-

tive except to prevent the psychological and physiological effects of inactivity.<sup>107-109</sup>

"We must tell our patients of the good prognosis following an attack of acute low back pain, we must instruct them in what to do, how to move and of the beneficial effects of mobilization towards work."<sup>102</sup> Exercises in this respect are a mandatory aspect of the treatment protocol.

Gradated, supervised, specifically prescribed exercises also allay the fear of recurrence from "any movement or activity."<sup>110</sup> The fear of recurrence from any activity is prevalent and must be addressed in treating the patient with acute low back pain. Exercise addresses this if there is concurrent reassurance and explanation by the physician in meaningful words and examples. The possible discomfort of exercise must be explained and justified.

Walking, as an exercise prescription, is probably the simplest, least stressful, and most beneficial therapeutic exercise.<sup>111-113</sup> This exercise requires no equipment but may be difficult to prescribe as more esoteric though unproven exercises for this poorly defined disease entity enjoy popular advertising and promotions.

**Specific Exercise Protocols.** Having discussed the principles of exercise and their documentation, it is still mandatory that the precise exercises advocated in the treatment of the patient with low back pain be discussed. As stated, there are various benefits from exercise that justify its implementation.

Flexibility, as a basis for exercise, enjoys unanimous acceptance among all specialties. What tissues are being stretched needs clarification. Muscles and their fascia needed repeated passive and active elongation to maintain their physiological length.<sup>114</sup> Tendons and ligaments are also in that category, as are the capsules of synovial joints. The collagen fibers are the basis of maintained elongation, as are fibrous and elastin tissues. Even the annular fibers of the intervertebral disk annulus, consisting principally of type I collagen fibers with an increasing percentage of type II fibers, are in need of physiological elongation for their nutrition.<sup>115</sup>

In a tight low back, the trunk does not fully flex either in forward flexion, lateral flexion, trunk rotation, or extension. In forward flexion, the physiological lordosis does not reverse into physiological kyphosis.

Limited flexibility may be the result of pain or may be the sequela of restricted physiological elongation of the soft tissue from disuse. Active flexibility as compared with passive flexibility may be difficult to differentiate. Fear of recurrence of low back pain from activity may be a factor in the inflexibility discerned during the examination.

Restoration of flexibility after an acute episode is usually attempted, along with a strengthening program. Each spinal segment



must be addressed, as also must be the pelvis and lower extremity. Proper spinal alignment demands total flexibility to the physiological limits of the component tissues.

When one appreciates the varied directions of the thoracolumbar fascia (see Fig. 1-47), it demonstrates the difficulty in ensuring adequate flexibility of all the soft tissues of the trunk.

In any treatment protocol, warm-up is considered beneficial before initiating exercise. Exercise often benefits from the application of heat or ice, depending on the indications.

Flexibility exercises aim at improving disk nutrition<sup>116</sup> and growth of the collagen fibers within.<sup>117,118</sup> Self-directed stretching and mobilization programs correct the fascial, muscular, ligamentous, and capsular shortening. As most of the motion (75%) of the lumbosacral spine occurs at L5-S1,<sup>119</sup> if that segment is restricted, it will cause excessive motion at other segments: in this case at L4-5 segments, with degenerative changes of these segments enhanced from increased shear.<sup>120,121</sup>

In total spinal flexibility the pelvis must be addressed to ensure a correct lumbar pelvic rhythm (see Chapter 1). Pelvic flexibility includes the glutei, hamstrings, and the gastroc-soleus, which must be elongated without increasing stress on the lumbosacral spine during the exercise. This indicates protective hamstring stretching (Fig. 10-19).

Flexibility and strengthening exercises also address restoration of proprioception.<sup>122-124</sup> Proprioception is the transmission of joint position information in complex activities.

Physical response of dense connective tissue to therapeutic stress is the basis of stretch exercises.<sup>125</sup> The immediate response to stretch may be a transient increase in tissue length, which indicates why exercise must be done repeatedly and consistently.

The responses of tissue to elongation activities are itemized in the following.<sup>125</sup>

1. Collagen fibers that are elongated 1% to 1.5% for less than 1 hour show no permanent deformation (elongation).
2. Elongation of 1.5% to 2% maintained for more than 1 hour will result in permanent elongation because that degree of stretch results in a melting of the tropocollagen bonds. The gain may be lost if the elongation is less than 1 hour or if the tissues are allowed to decrease their elongation.
3. Elongation of 2% may allow return to prestretch length if not followed by sustained or intermittent stretch during the subsequent 24 hours.
4. Elongation of 3% to 8% may cause a loss of continuity of the collagen and result in damaged or inflamed tissue.
5. Permanent stretching or excessive elongation tears the structure



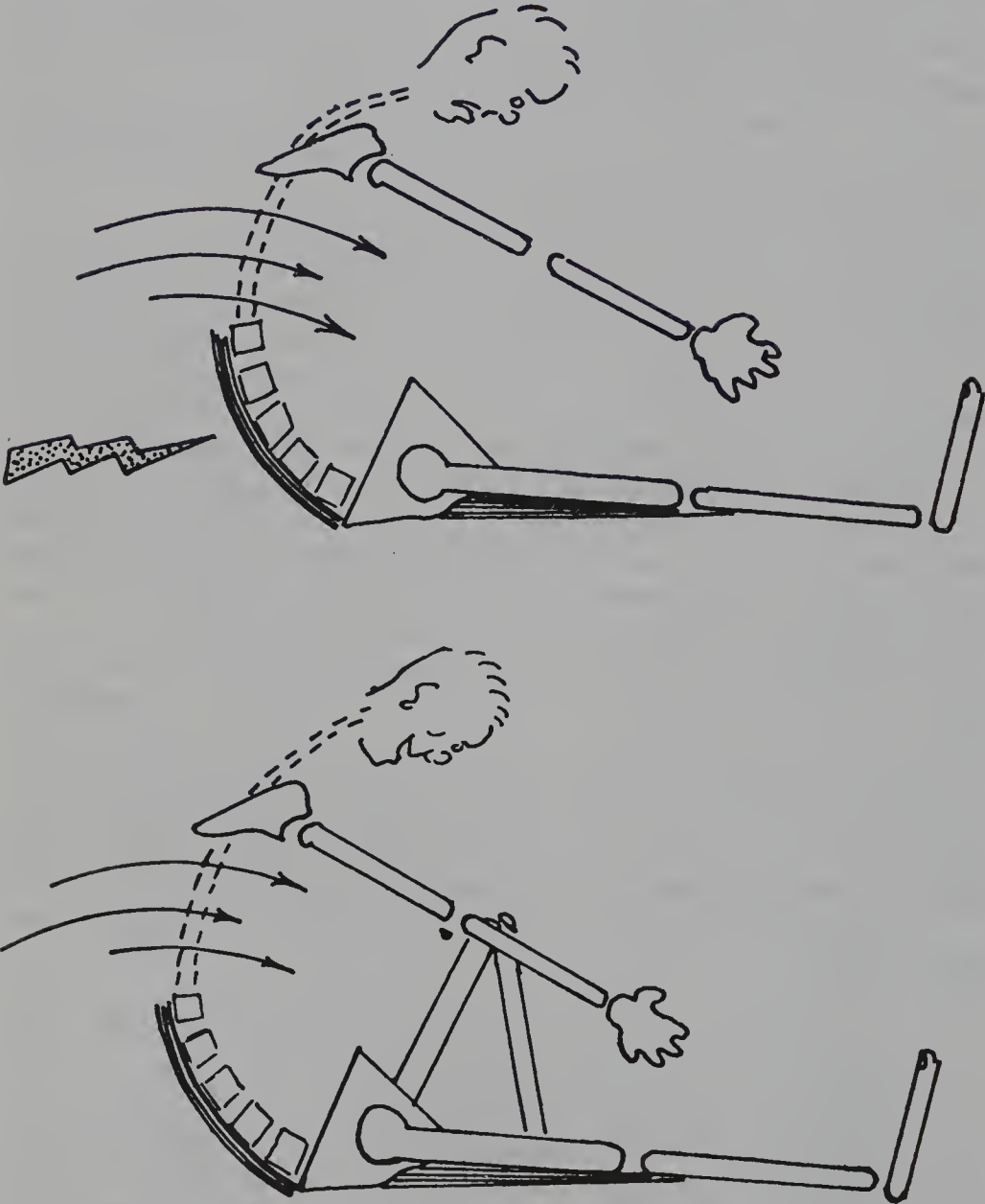


Figure 10-19. Protective hamstring stretching exercise. When the hamstring muscles are restricted in their elongation and considered to contribute to low back pain they can be gently stretched with the opposite leg flexed to avoid further low back pain that would occur if both legs were simultaneously stretched.

of the collagen fiber or disrupts the intermolecular bonds between the tropocollagen units, causing permanent damage to the collagen fibers(s).

Short-term strain of 4% to 6% causes a permanent increase in the length of the tissues by disruptive forces. Strains of 1.5% to 2% maintained for periods of longer than 1 hour will also destroy collagen. This indicates the need to be judicious in stretching efforts in clinical management of musculoskeletal conditions: in ligaments, in capsular and myofascial tissue, and in annular disk fibers.

Flexibility exercises should be done gently, repeatedly, eliciting minimal and self-limited discomfort with no effort to exceed full range of motion, as determined by the patient rather than by the therapist using physiological standards.

In summary, immobilization causes a significant loss of dense connective tissue strength, predisposing it to damage on ultimate elongation. Allowing tissue shortening for any length of time in a soft tissue injury is thus to be avoided. Excessive and prolonged stretching after injury and its immobilization must be judiciously applied. The biomechanical properties of collagen must be kept in mind when applying stretch exercises. Passive (done to) stretching is far more dangerous than active (done by) stretching.

Self-applied stretching exercises are thus the best prescription for the patient with low back pain.

Walking remains the best form of exercise (Figs. 10-20 and 10-21) because it addresses every aspect of body physiology.<sup>111-113</sup> All tissues are involved, ensuring their physiological elongation, including the annular fibers of the intervertebral disks (Fig. 10-22). These benefits are also enhanced by the cardiovascular pulmonary and psychological benefits of rapid walking.<sup>126-129</sup>

Active exercise to regain or maintain flexibility can be done daily without the use of mechanical equipment (Figs. 10-23, 10-24, and 10-25). These exercises stretch the lateral ligaments (Fig. 10-26) and the fascia of the erector spinae muscles.

Rotational exercises are also mandatory, providing they are within the elongation limits of the annular disk fibers. Because the collagen fibers of the annulus are oriented in different directions, only half of the fibers resist clockwise rotation.

Disks are stiffer in torsion than in bending.<sup>129</sup> During axial rotation, the increased stress in the collagen fibers raises the hydrostatic pressure in the nucleus pulposus, but this pressure is significantly less than occurs in bending.<sup>130</sup> When there is a combination of compression and torsion, the disks completely recover if rotation does not exceed 9°.<sup>131</sup>

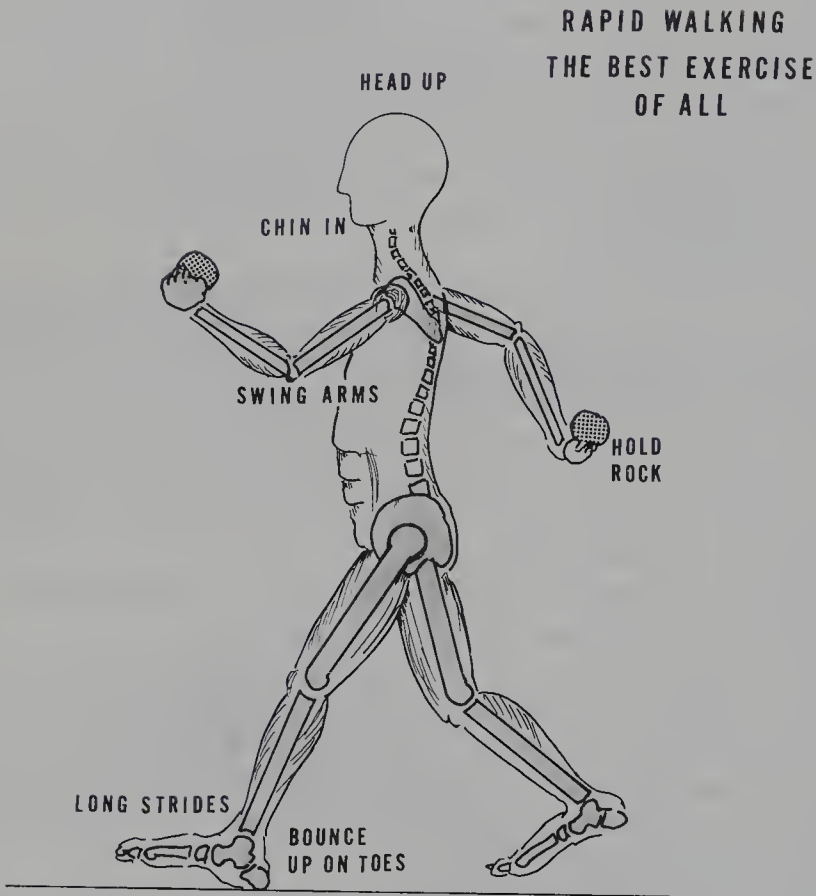
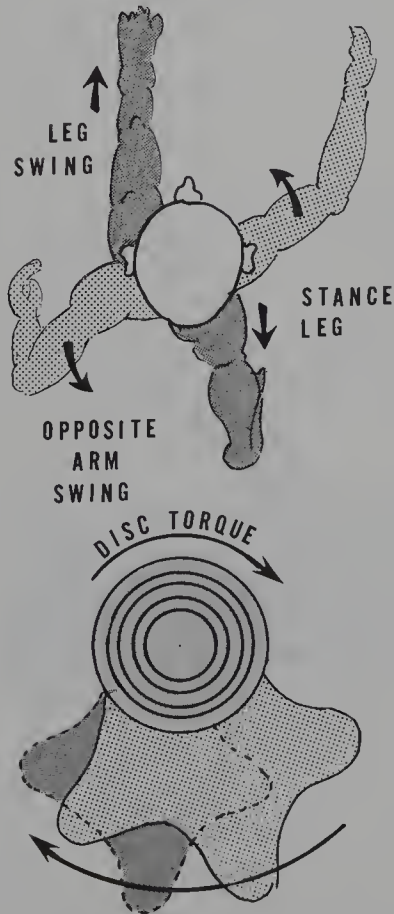
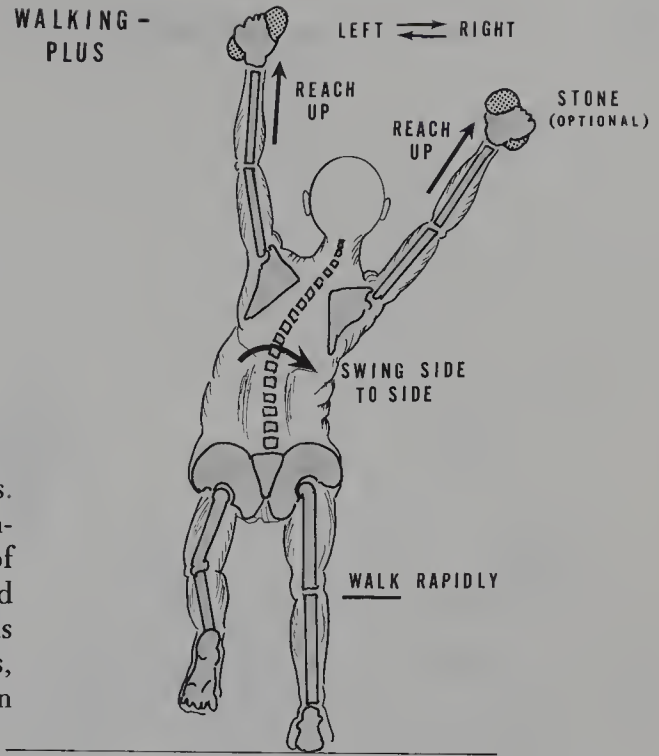


Figure 10-20. Walking: the best exercise. Walking has been accepted as the best comprehensive exercise. With swinging of the arms the trunk undergoes active flexion, gentle rotation, and lateral flexibility at each step. With attention, good posture is also enhanced. Holding a weight in each hand is optional. Besides trunk benefit there is no stress on the lower extremities and there is psychologic and cardiovascular benefit.

**Figure 10-21. Walking plus.** Walking as depicted here, increases active stretching of the lateral trunk muscles and also stretches and strengthens the latissimus dorsi muscles, which are muscles involved in proper back function.



**Figure 10-22. Disk torque in walking exercise.** Walking rotates the trunk gradually and gently stretches the annular fibers of the disk in a physiologic degree of elongation. This benefits the nutrition and stiffness of the disk.



Figure 10-23. Low back flexibility exercises. Exercises to elongate the soft tissues of the low back can be done simply at home, periodically during the day by laying supine and gently bringing both knees to the chest. The patient is “in charge” as to frequency and intensity, diminishing the fear of damage.

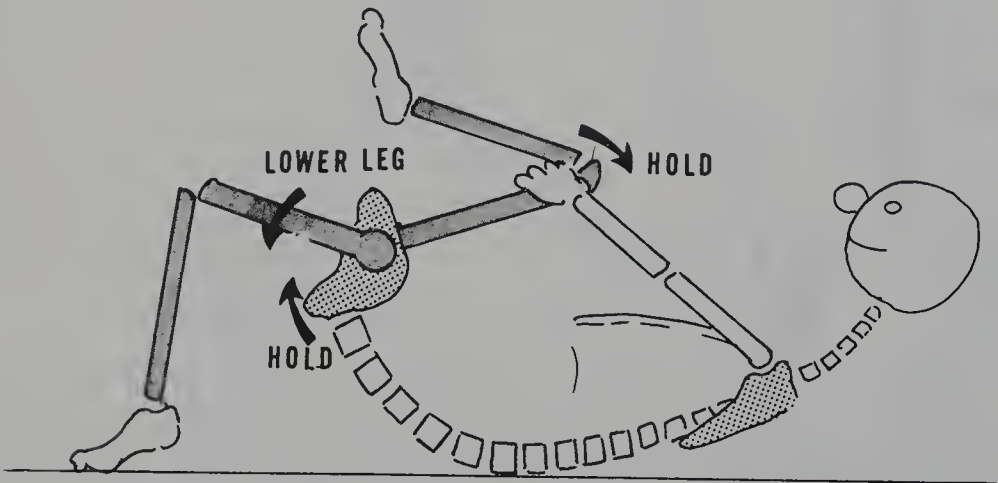


Figure 10-24. Descent from flexion exercise. After having flexed the low back with both legs to knees, one leg is gradually extended while the other leg remains flexed (knee to the chest). This stretches the hip flexors and maintains the flexed lumbosacral spine to avoid lumbosacral hyperextension when both legs are simultaneously extended.



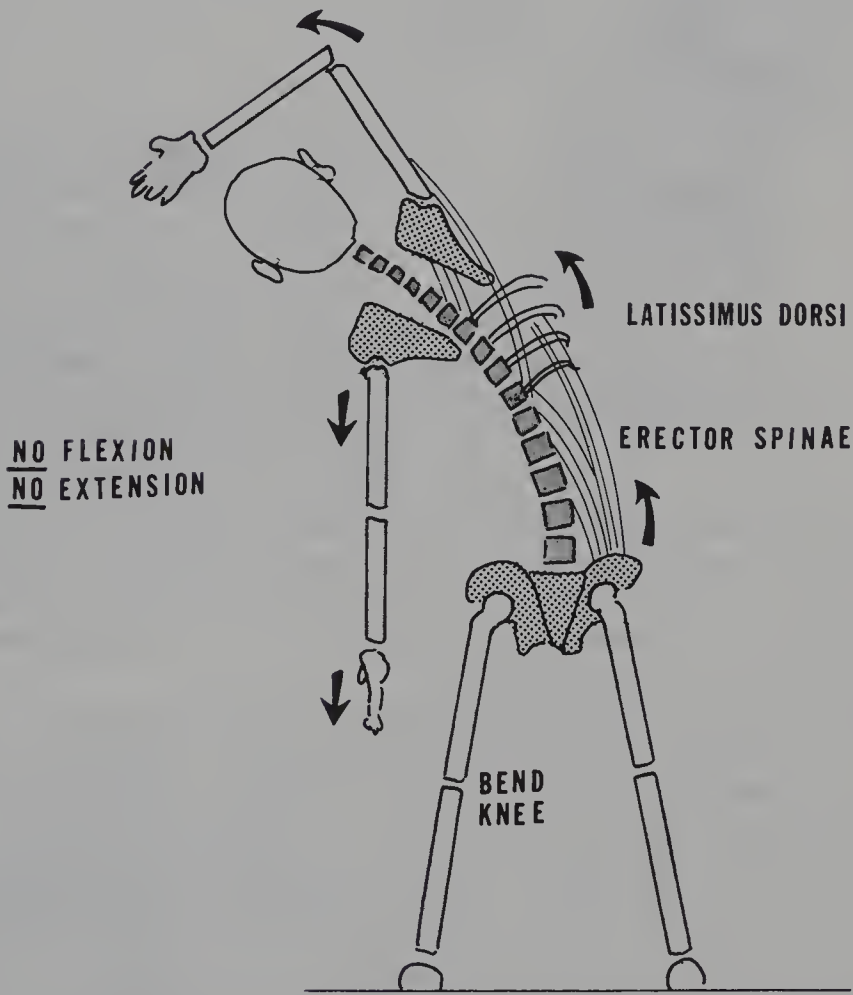
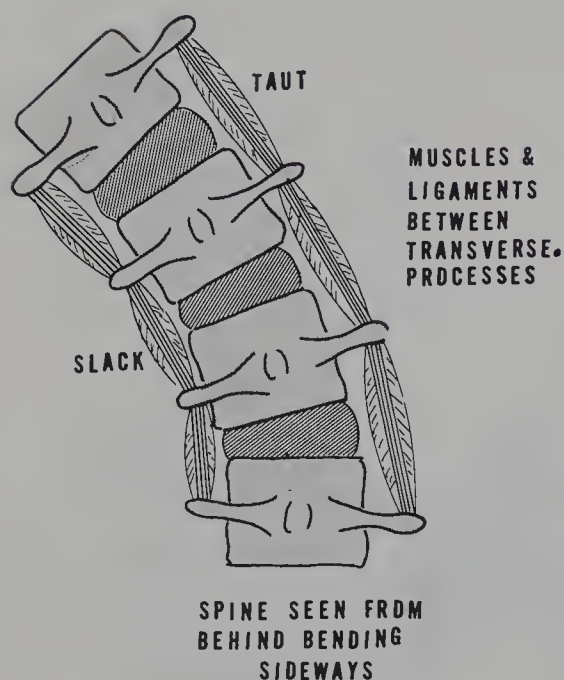


Figure 10–25. Lateral trunk flexibility exercise. With both feet slightly apart, which immobilizes the trunk, the upper body is gently and progressively bent lateral. With the arm overhead the latissimus dorsi is also stretched as are the paraspinous ligaments and fascia. Slightly flexing one knee prevents extension of the low back and is optional.



**Figure 10-26.** Lateral ligaments and paraspinous muscles of the low back. The tissues of the lumbosacral spine that are stretched in lateral flexibility exercises are depicted. (From Cailliet, R: *Understand Your Backache*, FA Davis, Philadelphia, 1984, p 131, with permission.)

Normal disks completely fail when rotation exceeds  $10^{\circ}$  to  $26^{\circ}$ . At failure, the outer lamellae of the annulus separate from each other and tear away from their endplate attachments. Torsion does not damage the nucleus or inner annular fibers and does not cause radial fissures, so it does not cause disk prolapse.<sup>132</sup>

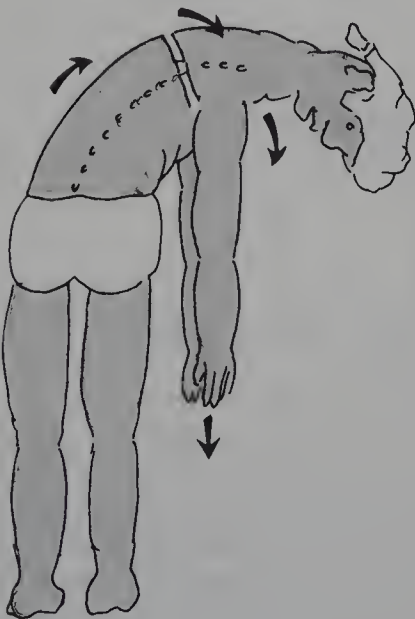
The lumbar apophysial joints limit axial rotation to about  $1^{\circ}$  to each side per lumbar level. Repeated small rotations of  $1^{\circ}$  could cause microscopic damage, but this has not been confirmed in normal disks.<sup>133</sup> Pathological disks are another situation.

Keeping in mind that rotational torque stresses have been postulated to stress annular fibers of the intervertebral disk, gentle rotational exercises (Fig. 10-27) are physiologically beneficial. As a combination of flexion and rotation occurs repeatedly in most activities of daily living, exercises to maintain this flexibility are of value (Fig. 10-28). These exercises also enhance the proprioceptive aspects of the motion. The exercises must observe flexion and physiological re-extension, which is mandated by proper body mechanics, as stated in Chapter 1.

Limited elongation of the hamstring muscles imposes excessive stress upon the lumbosacral spine in flexion activities. Exercises to elongate these muscles and their fascia must be undertaken repeatedly in a slow manner. These exercises must take into consideration the effect on the low back and thus be what can be termed unilateral "protective hamstring stretch exercises" (Fig. 10-25).



**Figure 10-27.** Rotational exercise. To maintain lateral flexibility of all paraspinous tissues, including the disk annular fibers, the person stands with feet slightly apart and rotates the upper body gently and rhythmically in both directions with increasing degree as tolerated.



**Figure 10-28.** Flexion rotational exercise. Because there are so many daily activities that require flexion and simultaneous rotation, an exercise that augments this motion is valuable. With both feet apart to stabilize the trunk the upper body is gently flexed and rotated to either side returning slowly and properly. This exercise also enhances the brain pattern that becomes implemented in daily activities.

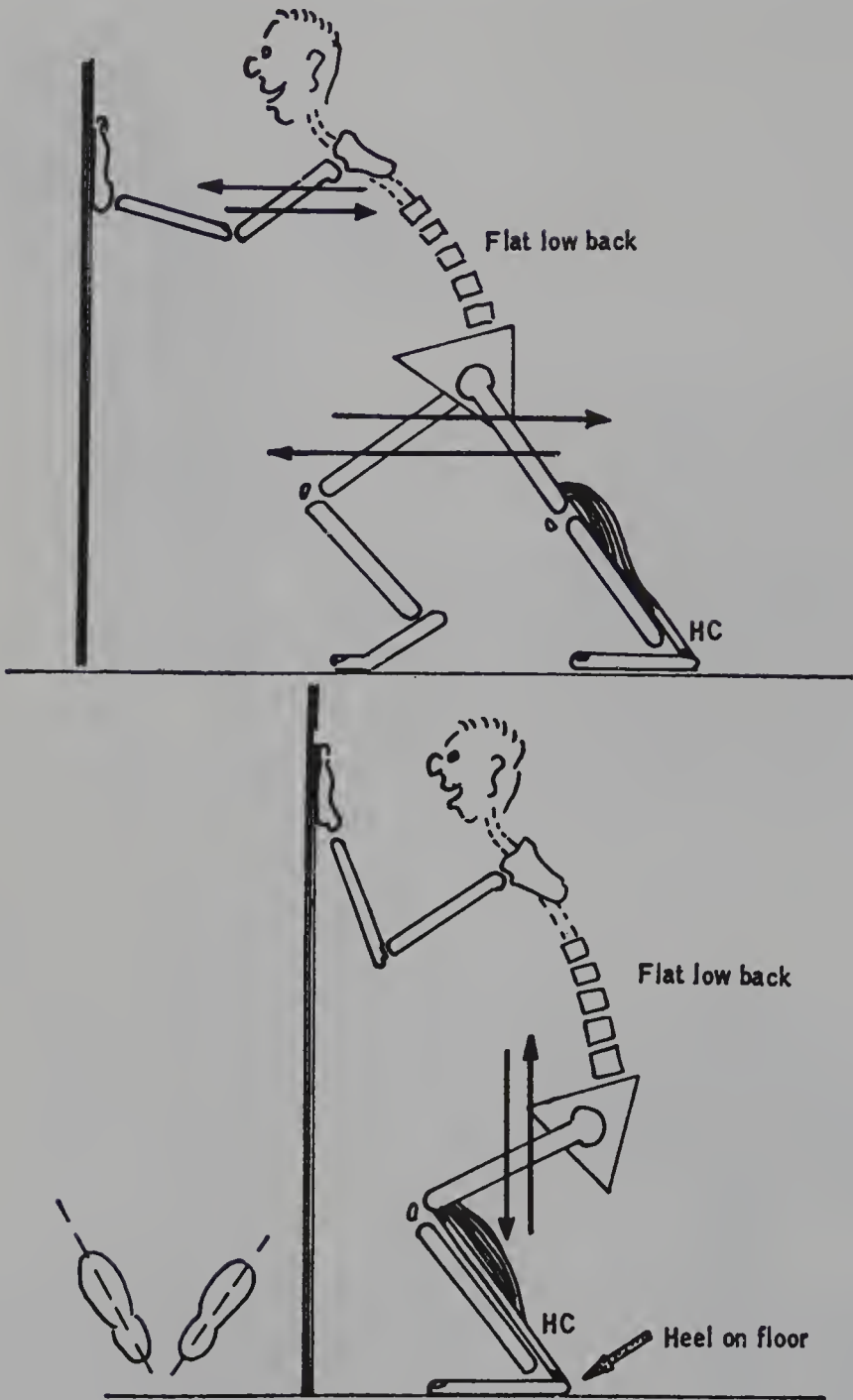


Figure 10-29. Heel cord stretching exercise. The *top view* depicts the person leaning against the wall that is significantly ahead of the center of gravity. One leg gastrocnemius muscle at a time is stretched by leaning forward keeping the foot firmly against the ground. Gentle rhythmic stretching is encouraged. The *bottom view* depicts stretching the heel cords while doing a deep knee bend and keeping both feet firmly on the ground. This exercise mandates that there is nothing wrong with the knee and that the deep knee bend is not too severe nor causing any discomfort.

As well as stretching the hamstrings, the heel cords (gastroc-soleus muscle tendon complex) must also be frequently stretched, as they impose limited total flexion as well as limited hamstring elongation (Fig. 10-29).

The hip flexors have also been impugned in causation of low back pain and should be evaluated and, if found limited, be elongated to their physiological limits (Fig. 10-30).

**Strengthening Exercises.** Strengthening exercises of the muscles of the trunk have been advocated by all therapists treating low back pain syndromes, even though their scientific justification remains questioned.

Strengthening of the abdominal muscles has been more clearly verified. Their role continues to be explored and explained. The concept of the "air bag" unloading the spine<sup>134,135</sup> was postulated as the basis for unloading the spine but was refuted as the needed intra-abdominal pressure exceeded the pressure within the abdominal blood vessels. A more acceptable concept is the effect of the oblique abdominal muscles on the thoracolumbar fascia, creating a compartment of the erector spinae muscles<sup>111</sup> (Fig. 10-23). The action on the fascia from the oblique muscles widens the fascia and creates a compartment tension that unloads the spine.<sup>136-141</sup>

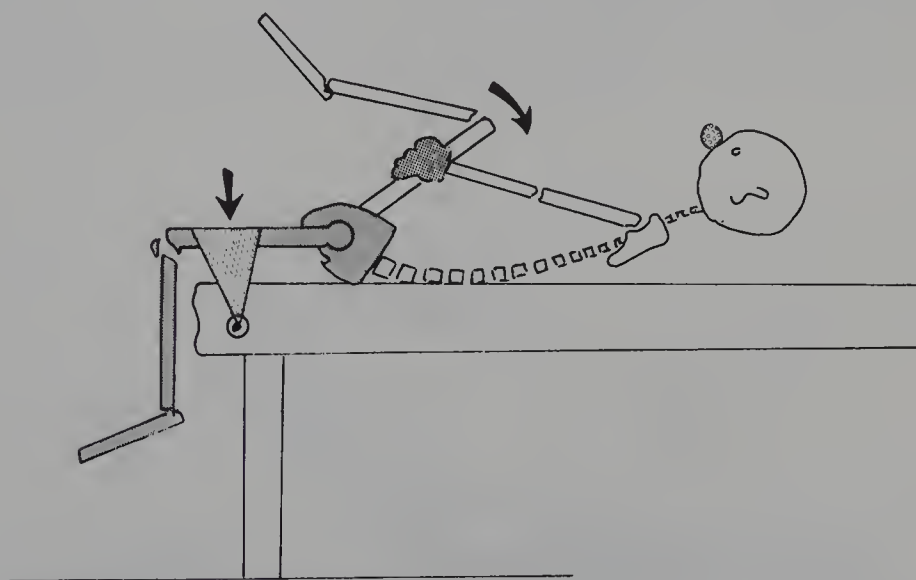


Figure 10-30. Hip flexor stretching exercise. This exercise requires equipment and some assistance and should only be considered when there is significant and pertinent hip flexion contraction. The hip to be stretched is firmly resisted by a strap to the table. The patient then applies stretch forces slowly, rhythmically, and progressively by flexing the opposite leg to the chest and holding it there briefly.



This concept implies the need to strengthen the oblique abdominal muscles as well as the sagittal flexors.

**Inappropriate Abdominal Flexor Exercises.** The traditional abdominal flexor strengthening exercises have been the sit-up, the sit-back, and bilateral straight leg raising. The last mentioned is appropriate only in people who have adequate abdominal muscles, as this exercise (Fig. 10-31) is often done with simultaneous hyperextension of the lumbar lordosis, which may cause discomfort (Figs. 10-32 and 10-33).

The ideal sit-up exercise to strengthen the abdominal muscles is performed with the legs flexed at the hips and knees. The exercise is done in stages. Initially the head is flexed, which involves the neck flexors. This initiates synchronous total flexors to contract. The next phase is to elevate the thorax and gradually progress to a total sit-up (Fig. 10-34).

Essentially a total sit-up is not necessary. Merely to elevate the thorax from the floor contracts the abdominals to a full contraction. The later sit-up stages become redundant.

The sit-back exercise begins with the person fully flexed both at the legs and the trunk and involves a gradual descent backward done in stages and degrees (Fig. 10-35). At any stage the body can be held, causing an isometric contraction, which adds endurance to strengthening. This exercise is ideal for the poorly conditioned, as it is easier and also addresses eccentric muscular activity.

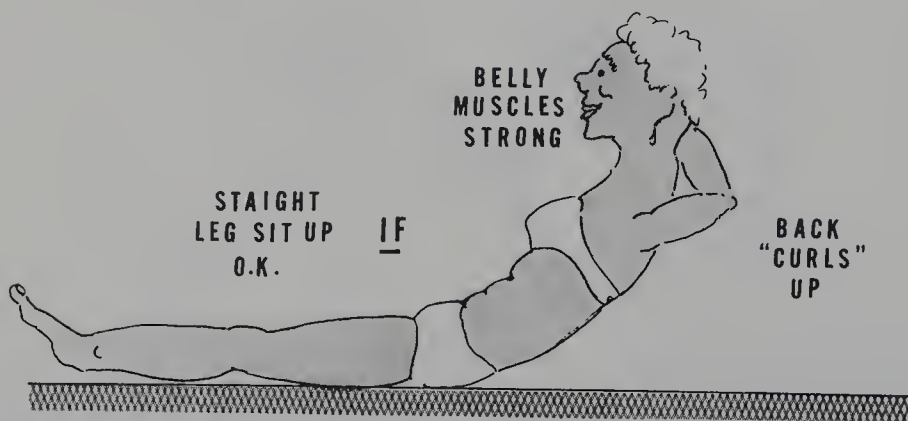


Figure 10-31. Sit-up with legs extended. Sit-up exercise with the legs extended to strengthen the abdominal muscles is appropriate if the person has reasonably strong abdominals and does not hyperextend the low back when performing the exercise. (From Cailliet, R: Understand Your Backache. FA Davis, Philadelphia, 1984, p 126, with permission.)

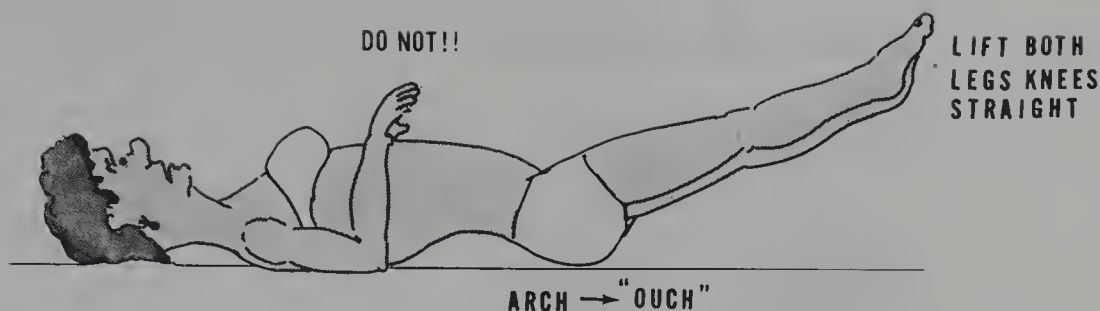


Figure 10-32. Inappropriate straight leg raising exercise. Bilateral straight leg raising from the supine position is not advisable if the abdominal muscles are not sufficiently strong enough to prevent the low back from arching during the exercise. In many people, hyperextension of the lumbosacral spine is symptomatic and in disk disease this exercise also increases intraspinal pressure (Valsalva effect).



Figure 10-33. Wrong abdominal strengthening exercise. Sitting up from the supine position with legs extended and arms behind the head, which increases the weight of the upper trunk, has the tendency to hyperextend the low back and may be detrimental. (From Cailliet, R: *Understand Your Backache*. FA Davis, Philadelphia, 1984, p 125, with permission.)

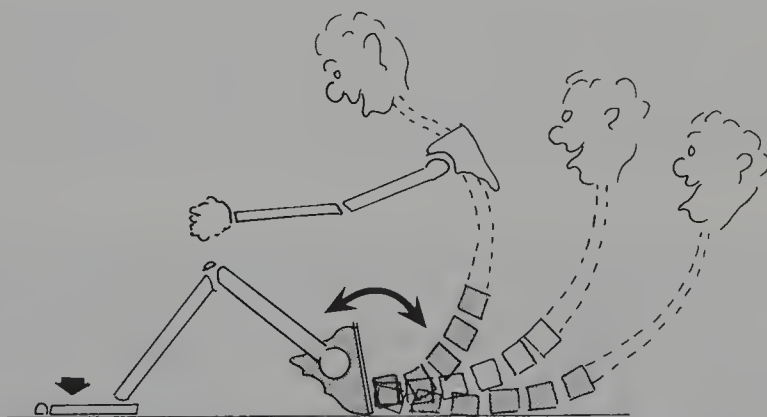


Figure 10-34. Abdominal flexion exercises: sit-backs. This exercise termed *sit-back* is a deceleration exercise preferable for people in relatively poor condition. Beginning at the total flexed seated position the upper trunk is gradually lowered and held then full sitting is resumed. Gradually the degree of sit-back is increased.

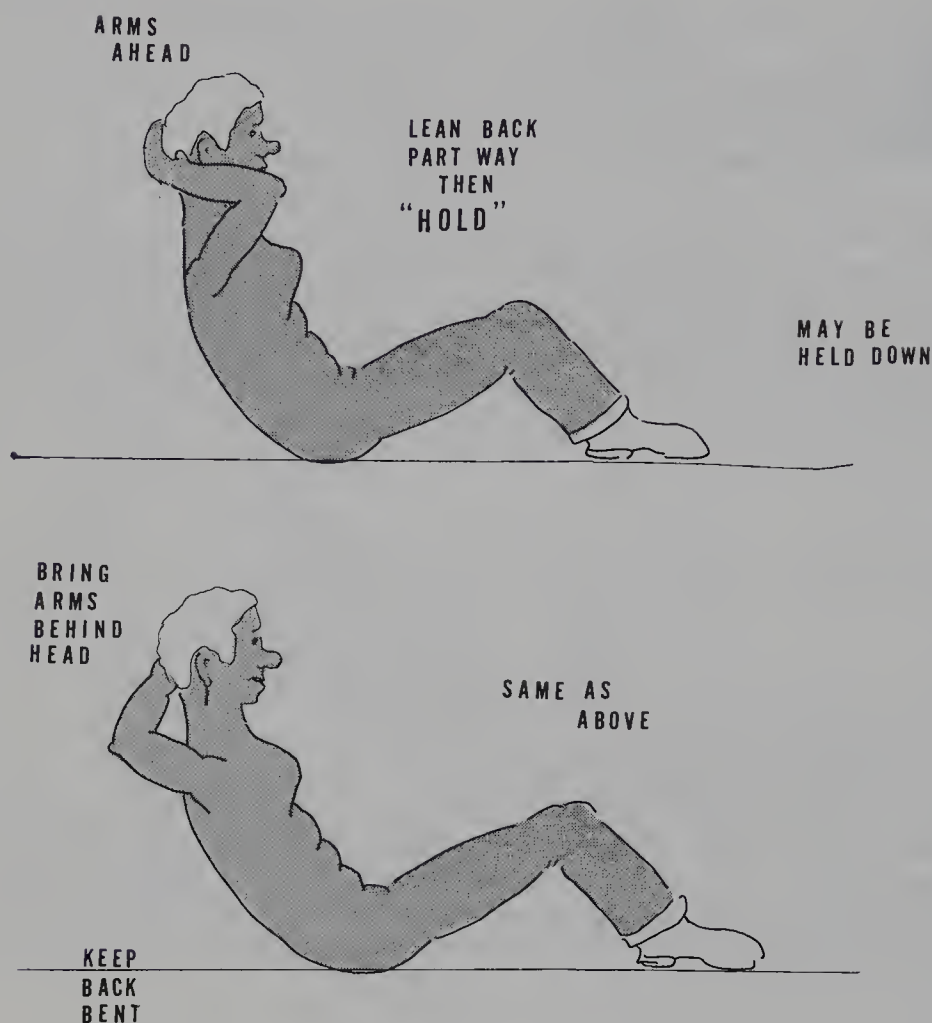


Figure 10-35. Enhanced sit-back exercise. The sit-back exercise described in Figure 10-34 is enhanced by varying the position of the arms behind the head. With the elbow further behind the head the weight of the trunk is greater, increasing the resistance to the exercise. (From Cailliet, R: *Understand Your Backache*. FA Davis, Philadelphia, 1984, p 123, with permission.)

Stress should be placed on strengthening the oblique abdominal muscles for the reasons already postulated. Exercises with trunk rotation strengthen the oblique abdominal muscles (Figs. 10-36, 10-37, and 10-38).

The purpose of strengthening abdominal muscles as well as unloading the spine is to decrease the lordosis and improve the posture. In this sense it not only improves the strength but also the kinesthetics of the movement. To decrease the lordosis, many advocate the pelvic tilt exercise, properly performed (Fig. 10-39).

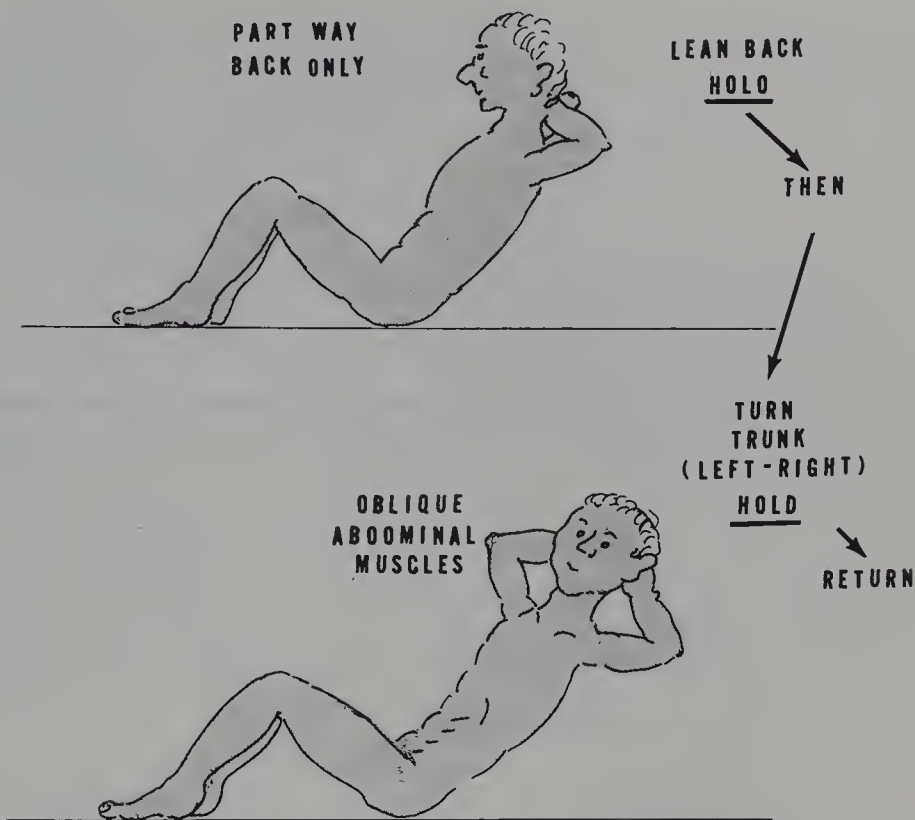


Figure 10-36. Oblique abdominal muscle exercise. Because the oblique abdominal muscles are considered most important in low back strength they must be strengthened. This exercise begins as a sit-back exercise with the exception that the trunk is rotated in both directions and held followed by appropriate return.

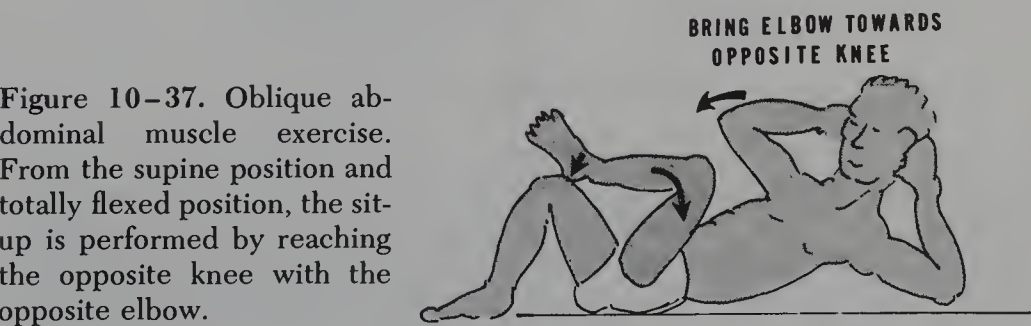


Figure 10-37. Oblique abdominal muscle exercise. From the supine position and totally flexed position, the sit-up is performed by reaching the opposite knee with the opposite elbow.

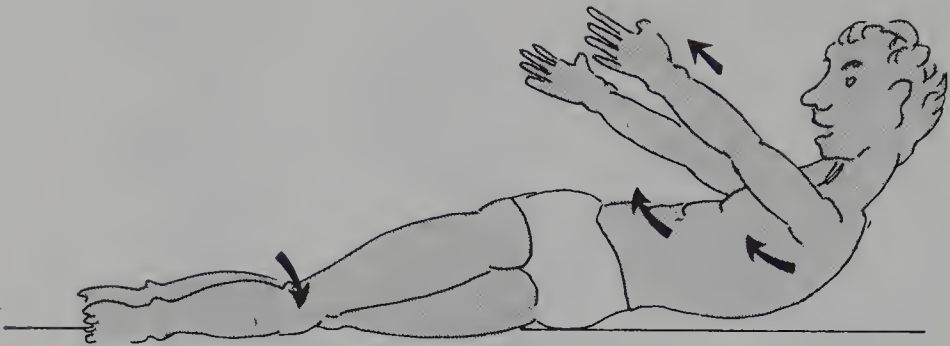


Figure 10-38. Oblique abdominal muscle exercise. From the supine position one leg is crossed over the other and the upper trunk is flexed in the opposite direction.

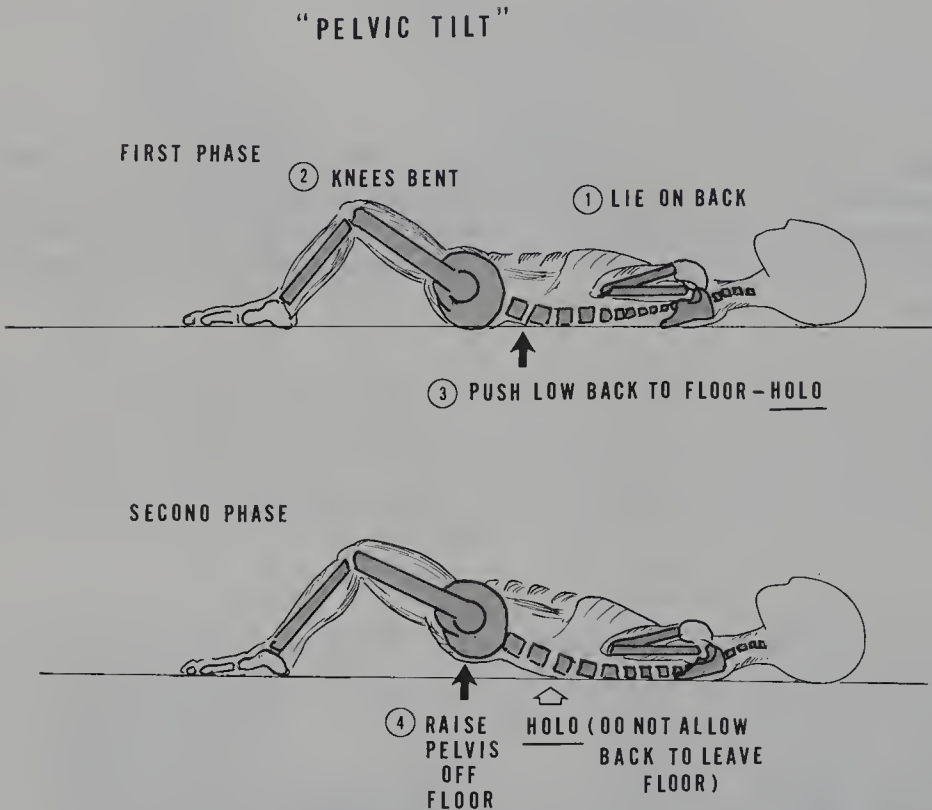
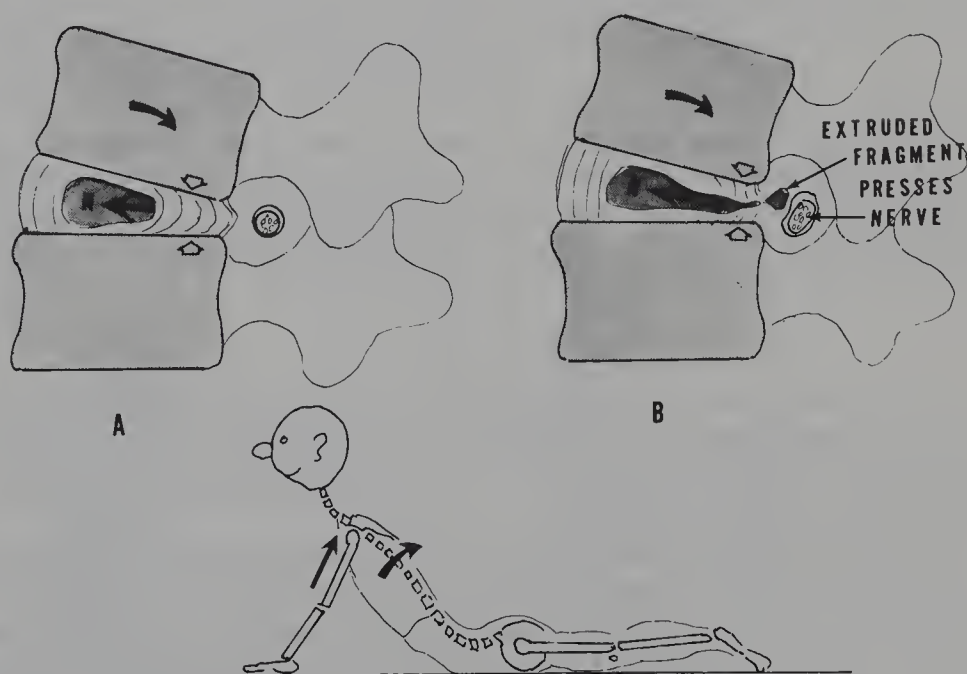


Figure 10-39. Pelvic tilt exercise. The classic pelvic tilt exercise develops a person's muscle strength and flexibility and decreases the risk of lumbar lordosis. From the total supine position (1) with the knees flexed (2) the low back is forced against the floor and held there (3). The pelvis is then slowly and progressively elevated from the floor (4) while the low back is held to the floor.



Based on the conclusion that annular disk tears occur from combined rotational, flexion, shear, and compressive forces, exercises implementing these actions must be done slowly, gradually, and progressively and always under careful control. Torsional exercises improve torsional strength and proprioception. When these exercises are performed with concentration and within physiological limits they minimize potential injury from inadvertent actions of bending and lifting.

In the treatment of alleged lumbar disk herniation as the cause of low back or leg pain, flexion exercises have been widely advocated. The concept of flexion exercises as opposed to extension exercises was challenged by MacKenzie,<sup>142</sup> who postulated that the disk nucleus could migrate anteriorly and posteriorly within the intervertebral disk by various spinal movements. Of special interest was that the posteriorly protruding nucleus against the posterior annular fiber protecting the posterior longitudinal ligament and the emerging nerve roots could be caused to migrate anteriorly and thus away from nociceptive tissues (Fig. 10–40). Treatment of a disk herniation was thus postulated to benefit from extension position and exercise (Fig. 10–41).



**Figure 10–40.** Provocative nuclear extrusion test. By assuming a passive hyper-extension position from the prone position, the nucleus of the intervertebral disk migrates anteriorly, away from sensitive tissues such as the posterior longitudinal ligament and the nerve roots. (A), It is a diagnostic and therapeutic exercise if done properly. If the disk has extruded, the test will not be confirmatory and the position not therapeutic (B).

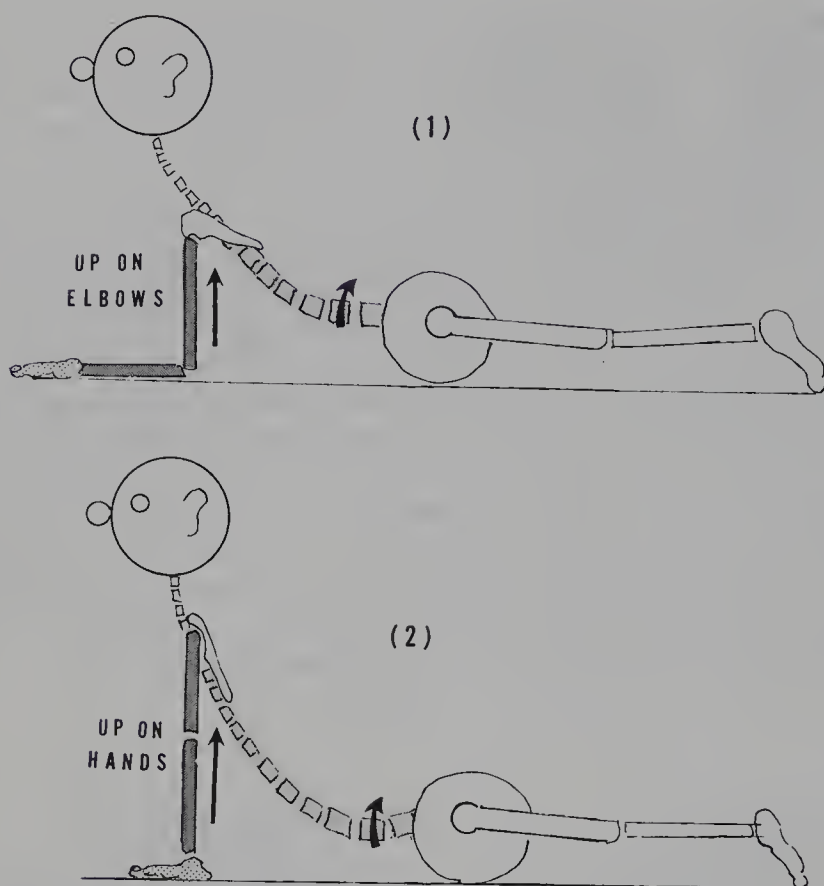


Figure 10-41. Provocative disk herniation test position. To perform the provocative disk herniation test properly the patient assumes the prone position and slowly elevates the upper portion of the body, using the arms only until the weight is borne on the elbows (1) then the arms are extended (2). All the efforts are from the arms not the low back extensor muscles.

The value of the pelvic tilting exercise, admittedly a flexion exercise, is not diminished by the MacKenzie concept as it is implemented after anterior migration of the nucleus but extension has been considered to have occurred.

The physiological basis for pelvic tilting has not been confirmed, but it probably regains lumbosacral flexibility through a limited physiological range and strengthens the abdominal muscles as well as the gluteals, which are accepted as being involved in spinal function. The beneficial decrease of lordosis is all that is questioned.

All the preceding exercises are useless if the body is inappropriately and incorrectly used. It is in faulty use or excessive use that the back fails and symptoms and pain occur.

The back school concept evolved from acceptance of this. The school aspect emerged in that a person can be taught how to stand

properly, bend properly, and lift properly.<sup>143,144</sup> *Properly* implies the procedure being physiological and under the conscious or subconscious control of the person. Normal actions are engrammed within the cerebral cortex as stated in previous segments of this text and can therefore be influenced by training, practice, and enforcement.

Farfan<sup>145</sup> stated, "It is very interesting how many interested men . . . do not agree that back pain is preventable or even controllable with back school methods. Many back schools . . . are simply exercise programs and audiovisual programs." He further defined back schools as "education and training . . . using the principle of body mechanics and back health care for prevention and control of back pain in the most efficient manner." He further postulated that "many back schools take on the behavioral approach to chronic disability . . . which is valid . . . as long as the behavioral approach operates under the umbrella of medical diagnosis and associates with structural disease care." This last statement is the concept that underlies the purpose of this text.

Ergonomics have also held prominence in the concept of proper body mechanics (Figs. 10-42 to 10-46).<sup>120,121</sup>

Because many injuries to the low back come from a lifting activity, proper bending and lifting are major subjects taught in the back school (Figs. 10-47 to 10-50). Improper bending and lifting are also discussed (Figs. 10-51 and 10-52).

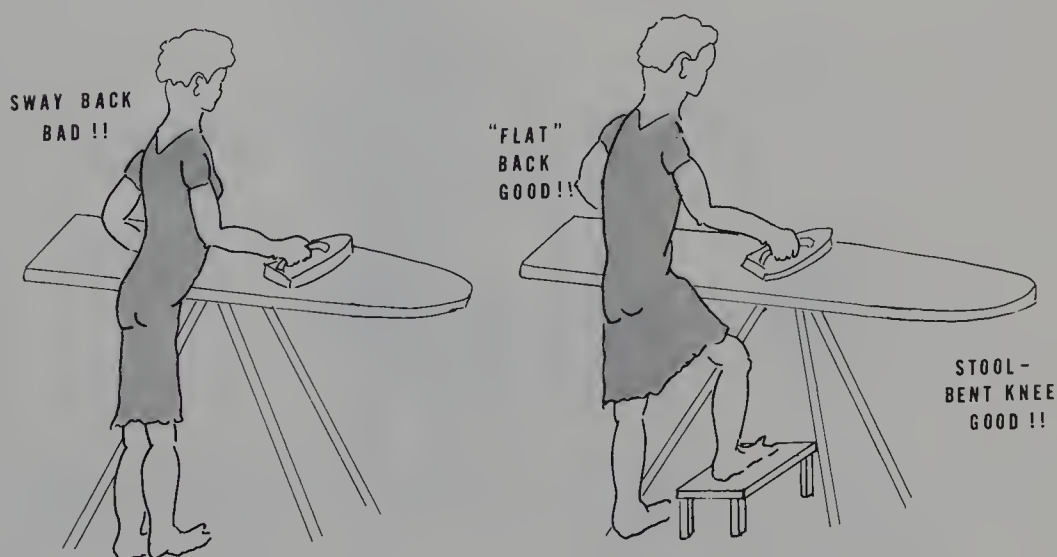


Figure 10-42. Proper prolonged standing. Prolonged standing with excessive lordotic posture may be stressful. Standing with one foot on a stool, which flexes the leg, causes the low back to decrease the lordosis. (From Cailliet, R: *Understand Your Backache*. FA Davis, Philadelphia, 1984, p 56, with permission.)

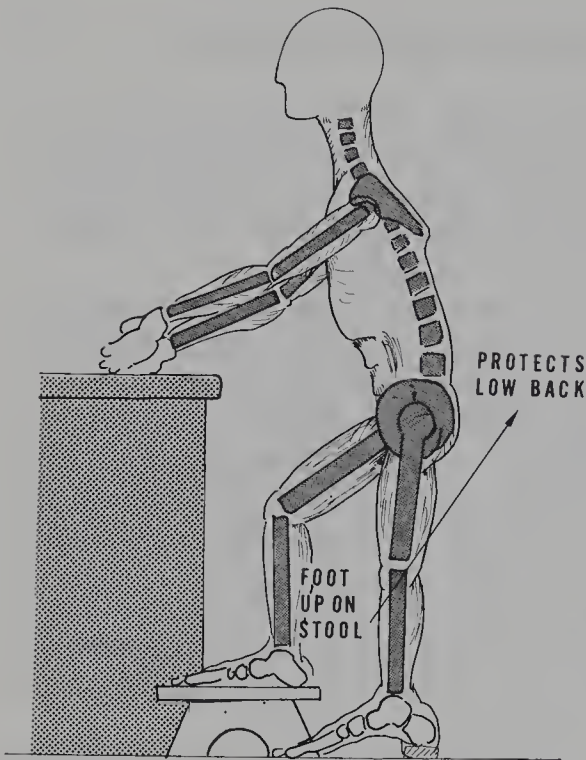


Figure 10-43. Proper standing posture to avoid stress. Prolonged standing with one foot on a stool protects the low back.

## IDEAL SITTING

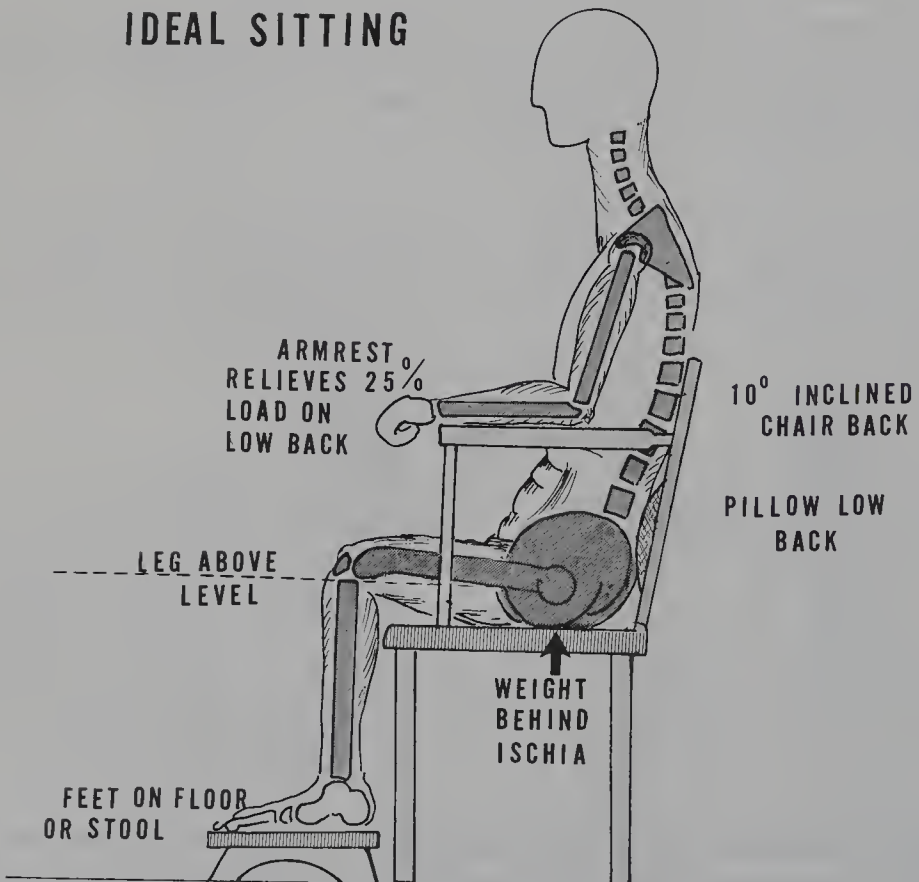


Figure 10-44. Ideal sitting posture. The chair should be high enough to allow the thighs to be horizontal and the feet on a firm surface. A slight lordosis is physiologic.

Figure 10-45. Proper one arm lift. One arm lift is demonstrated. (From Cailliet, R: Understand Your Backache. FA Davis, Philadelphia, 1984, p 144, with permission.)

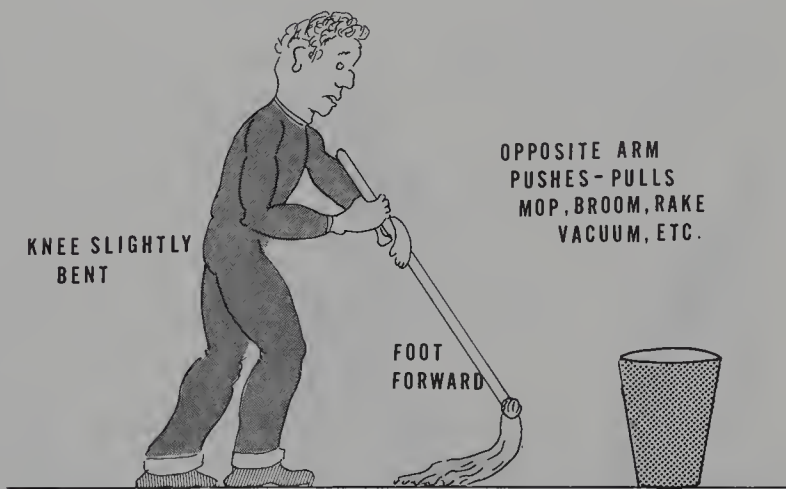
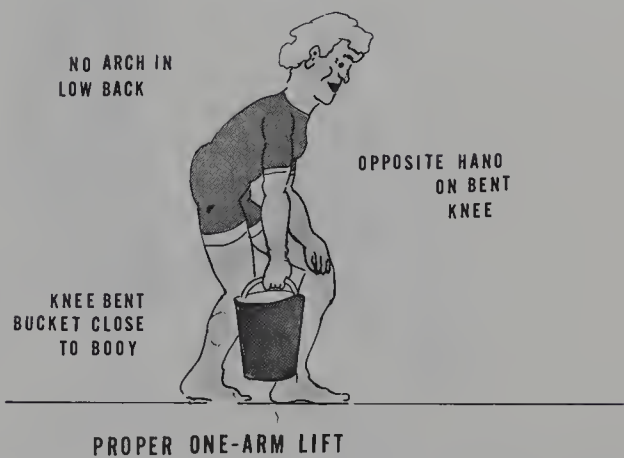


Figure 10-46. Diagonal principle of physical efforts. The diagonal principle applies to mopping, vacuuming, raking, and other similar activities.



## LIFT PROPERLY

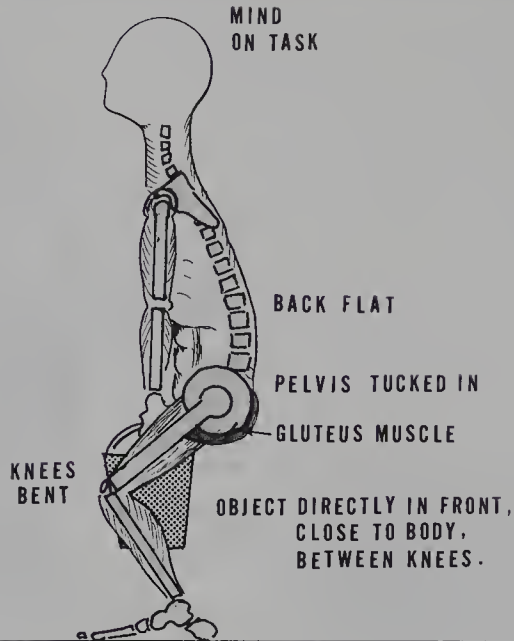


Figure 10-47. Lifting properly. All aspects of lifting properly are enumerated in this illustration. Most important is having one's mind on the task. Proper training makes the act automatic if practiced.

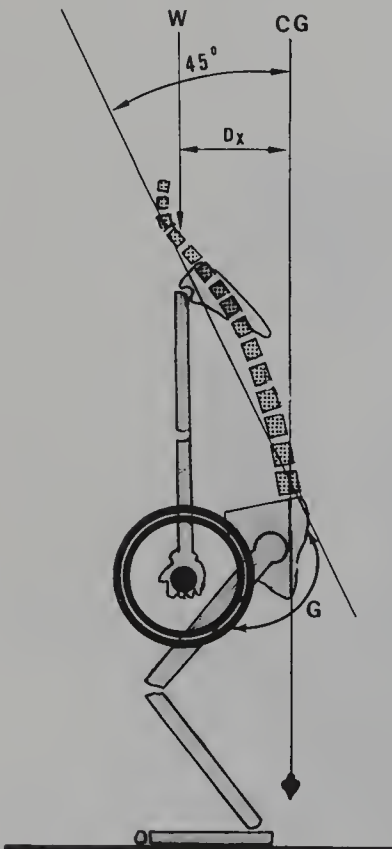


Figure 10-48. Analysis of proper lifting technique. With the center of gravity (CG) being the basis for this technique, the object lifted (W) should be close to the body ( $D_x$ ), the knees bent, and the pelvis tilted by the gluteal muscles (G). The spine is preferably at a 45 degree angle from the center of gravity.

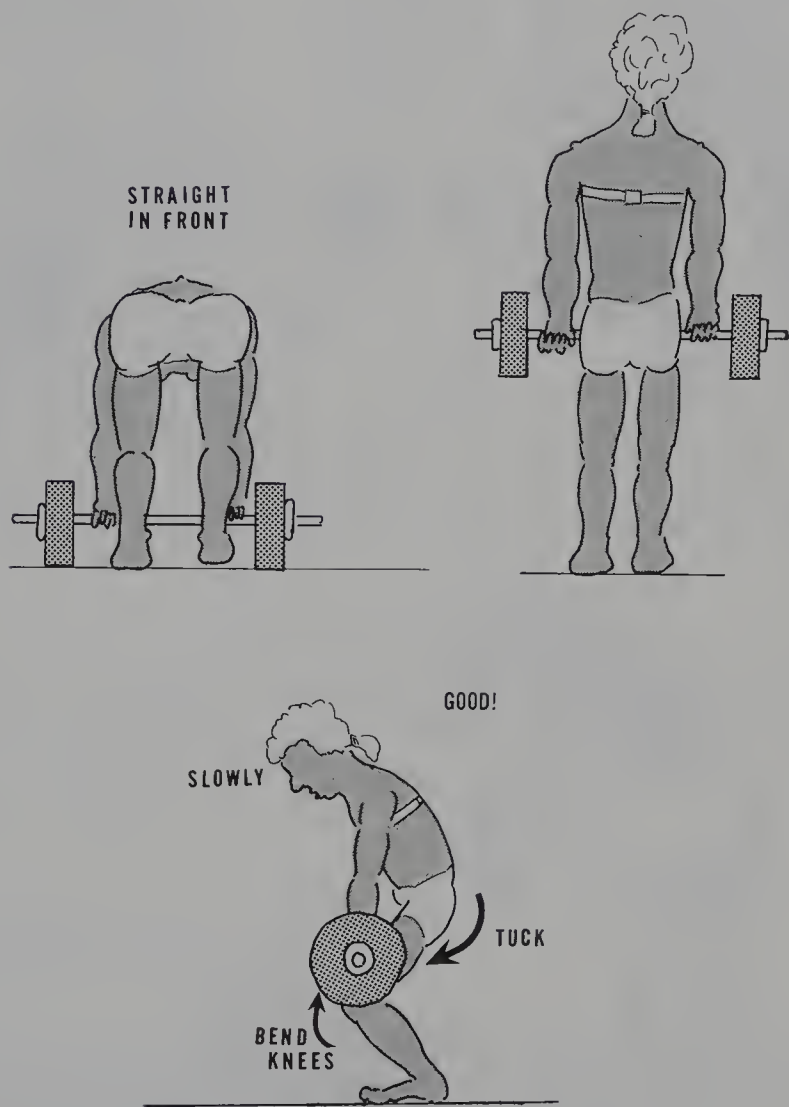


Figure 10–49. Proper manner for lifting a heavy object. All three views show the proper technique. The object is directly in front, picked up with knees bent, object close to the body, and the pelvis tucked in. The lift is primarily with the legs until 50% of extension has been reached.

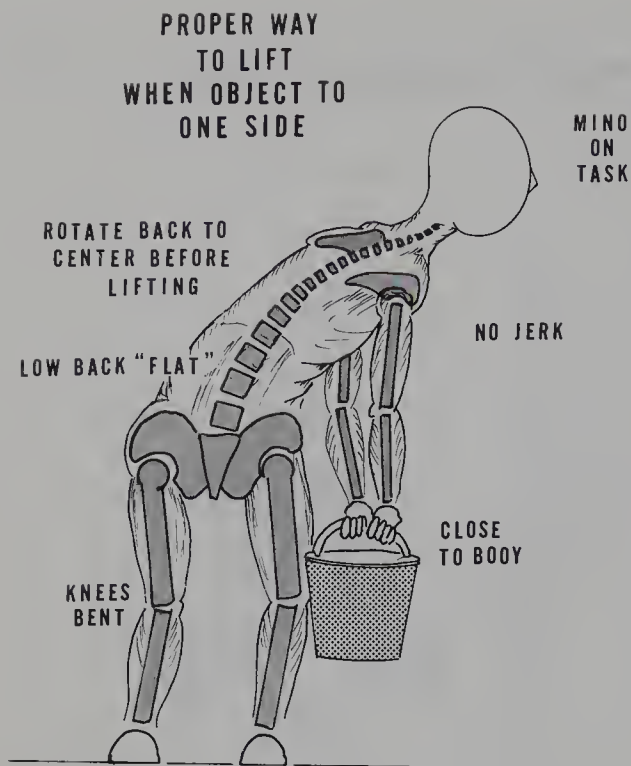


Figure 10-50. Proper way to lift an object to one side. The aspects of lifting are depicted. When the object to be lifted is to one side of the body. Proper derotation is mandatory. By repeated practice of this technique the probability of a faulty lift is minimized.

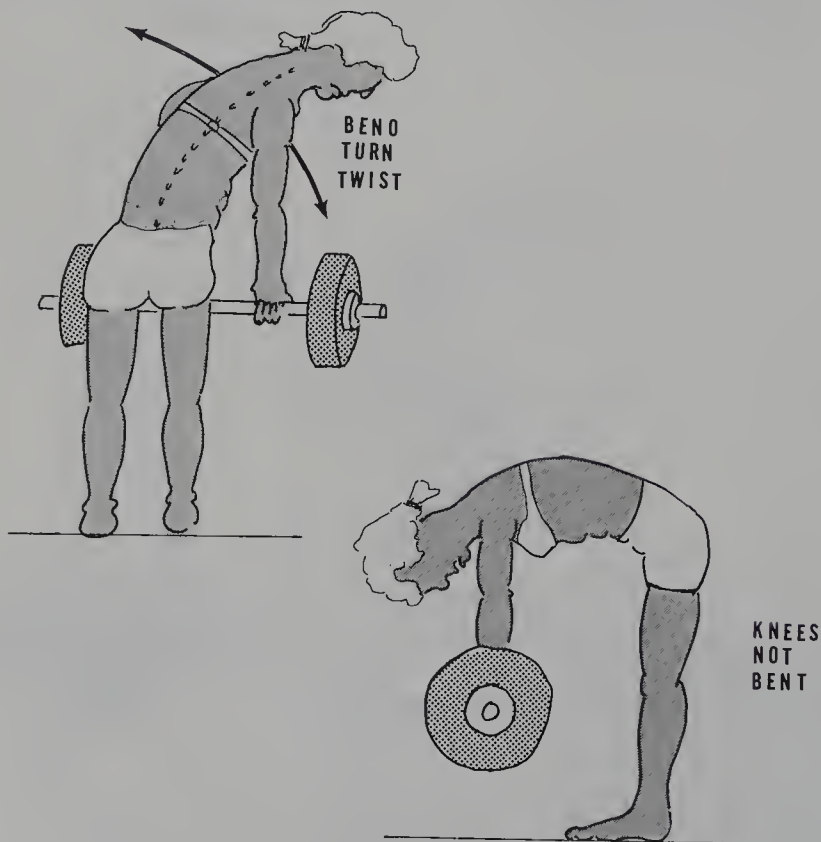


Figure 10-51. Improper lifting technique. Lifting with the knees straight must be avoided as it places the stress on the spine.

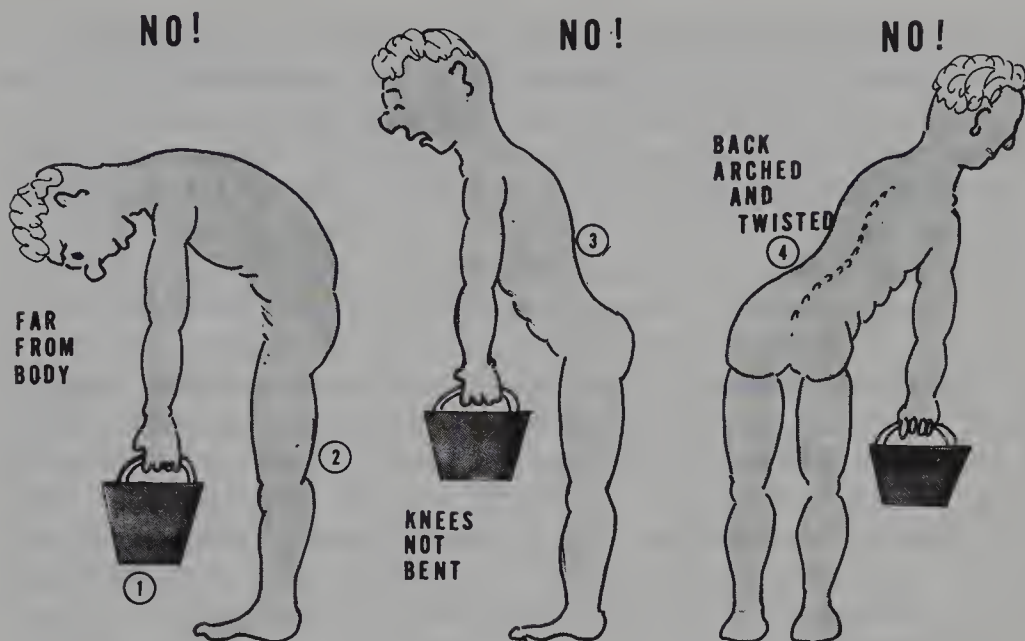


Figure 10-52. Improper aspects of lifting. (1), The object to be lifted is too far from the center of gravity; (2), the knees are not bent; (3), the low back reextends prematurely; and (4), the reextension does not derotate properly.

Farfan<sup>145</sup> wrote, “The subject matter . . . of the school must be tailored to the specific needs of the disabled.” This statement must be modified to include the specific needs of the potential victim. All activities of an individual make subject matters in any school a large agenda that must be addressed.

## Conservative Care of the Herniated Lumbar Disk

Voluminous literature is presented daily on the care of the patient with herniated lumbar disk yet in the comprehensive text *Contemporary Conservative Care for Painful Spinal Disorders* edited by Mayer et al. and written by numerous experts in the field, representing every specialty, only one significant mention is made for conservative nonoperative treatment,<sup>50</sup> with one reference<sup>57</sup>.

White,<sup>50</sup> in this article, states, “In summary, surgery for lumbar spine pain or low back pain with radiating leg pain has a notoriously unsuccessful track record. . . . Studies have shown that time alone may cure herniated disks for which surgery has been indicated.”<sup>50</sup> And McKenzie<sup>146</sup> writes, “Other studies have shown that aggressive conservative care (aside from time alone) returns as many as 92% of pa-

tients with herniated disks to normal activities within a few months.” In this excellent text, these “aggressive conservative treatments” are not specified other than by McKenzie,<sup>146</sup> who postulated that the herniated disk nucleus without extrusion could be physically returned to a more anterior position (Figs. 10–46 and 10–47) by a passive hyperextension position.

In summary, it can be stated that the occurrence and recurrence of acute low back pain and the prevention of chronic low back pain can be addressed by many modalities directed to diminishing pain and regaining good body flexibility, strength, and endurance as well as ensuring good body mechanics. The cause of low back pain remains mechanical in most cases, beginning with inappropriate movements, which result in pain and thus leads to disuse, disability, drugs, and depression. The tissue sites of resultant pain have been addressed and the plea remains that the prescribed treatments be meaningful, appropriate, physiological, and adequately evaluated to avoid excessive, prolonged, and non-beneficial outcomes.

## Manipulation

Manipulation has enjoyed advocacy in the treatment of low back pain problems that must be considered in the realm of treatment protocol.<sup>147–149</sup> There are many practitioners who claim that manipulation of the spine “unlocks,” mobilizes a “jammed facet,” reflexively releases muscle spasm, elongates the facet capsule, or realigns a “subluxed joint.”

So-called manipulation varies from gentle stretching to forceful application of manual force. The latter is *manipulation* and the former is termed *mobilization*. In many regions the latter can be administered only by a physician and the former is in the domain of the physical therapist.

The benefits from manipulation have been postulated to include:

1. A facet becomes immobilized by an acute synovial reaction, and adherence of the adjacent facet joint surfaces from an inappropriate motion or an abnormal external force. Manipulation separates these surfaces.
2. A meniscus that normally exists within the facet joint becomes entrapped from an unphysiological motion.
3. The redundant facet capsule becomes lodged between the adjacent articular surfaces.
4. The mechanoreceptors of the joint capsule are desensitized by an abrupt unphysiological motion of the joint, preventing further motion.



5. The spindle system of the involved muscles is impaired by an unphysiological motion. Manipulation allegedly reflexively stimulates and reciprocally relaxes the extrafusal muscles.
6. The involved spinal segments become malaligned by an unphysiological movement and are realigned into physiological position by manipulation. This explains the term *adjustment*.
7. Manipulation is a placebo that benefits the patient by a “laying on of hands.”

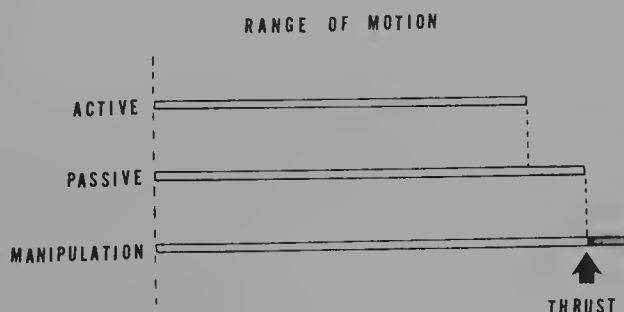
The physiological movements of joints are limited in their range by the elasticity of the capsules, the periarticular ligaments, and the fascial limits of the contiguous muscles. Joints have an active and a passive range (Fig. 10-53).

Exactly what occurs from manipulation remains conjectural. It affords benefit in many patients, but the basis for the improvement gained is obscure. The lasting benefits also have not been confirmed. The numerous techniques of manipulation have been well documented and will not be discussed in this text.

Manipulation is usually a force applied in the direction of restriction to regain that lost motion. Manipulation in the opposite direction, away from the locked position, has been advocated by Maigne.<sup>150</sup> A critical evaluation of manipulation is that it is often applied in a total manner, without specifically designating a precise functional unit that is allegedly locked. The long-arm technique is gross rotatory motion of the entire lumbosacral spine and the short-arm technique is aimed at a precise segment, but how this locale is determined remains unstated.

The concept of manipulation is to regain mobility, but it has also been advocated as improving the stability of a joint by realignment to its physiological position. *Clinical instability* is defined as “the loss of the ability of the spine under physiological loads to maintain relationships

Figure 10-53. Concept of manipulation. A joint has an active range of motion that can physiologically be exceeded passively. When that range of motion has been reached a firm passive thrust exceeds that range. The benefit of manipulation is claimed to be to regain the physiologic range that has been lost from pathology.



between vertebrae in such a way that there is either damage or subsequent irritation to the spinal cord or nerve roots . . . and development of incapacitating deformity or pain from structural changes."<sup>151</sup>

How instability of a spinal segment is clinically determined does not have a clear or valid definition. Even radiological studies fail to ascertain this instability. The role of muscles in clinical stability also remains obscure. The ligaments, joint capsules, and disk annular fibers admittedly afford stability.<sup>152</sup> The posterior longitudinal ligament is less important than is the anterior longitudinal ligament. The interspinous ligaments do not appear to be significant, but the facets play a critical function in spinal segment stability.<sup>152</sup>

Whereas most joints of the spine are incongruous (Fig. 10-54), they do not have intrinsic stability.<sup>153,154</sup>

The potential nerve damage from an unstable spinal joint in the lumbar region is less ominous because the cord is not present in the spinal canal and damage merely results in a nerve root loss.<sup>155</sup>

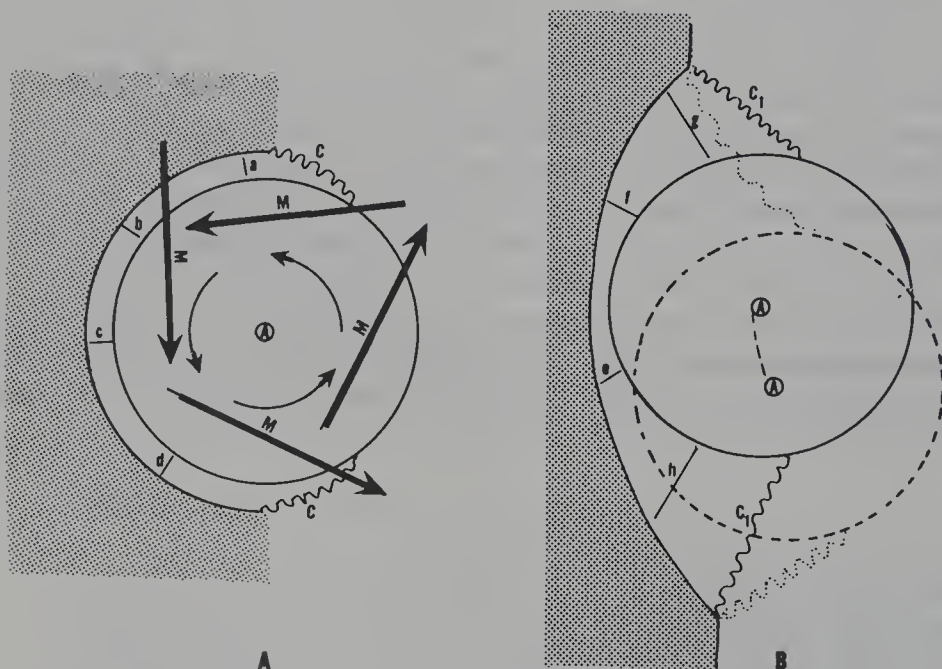


Figure 10-54. Congruous-incongruous joints. (A) A congruous joint where the concave and convex surfaces are symmetrical. The axis of rotation (A) allows the muscles (M) to rotate in a circular manner. The joint space is equal around the entire convex surface (a,b,c,d). The capsule (C) is equal on both sides. (B) An incongruous joint has asymmetrical surfaces of different curvatures, causing a difference in the joint space (e,f,g,h), a shifting axis of rotation, shearing movement of the two surfaces (*solid circle* compared with *dotted circle*), and the capsule C1 differs in its length.

A comment on the modality of manipulation is that the basis for it is unproved and that it is a passive therapy with no patient assistance. Long-range benefits (outcomes assessment) need confirmation. Its immediate benefit has been claimed and this cannot be disproved, though it is subjective. The prolonged and repeated application of adjustments with recurrence of the pain and disability is to be deplored and avoided.

## Traction

Traction has been used since the time of Hippocrates yet remains unconfirmed as to efficacy. Traction has enjoyed a range of evaluations, from being a specific modality for the relieve of mechanical low back pain and reduction of intervertebral disk protrusion to being merely a means of keeping the patient off his or her feet. The precise physiological basis for the benefit gained by traction remains controversial, as does the specific manner of its application.

Traction applied to the patient in the reclining position decreases the effects of gravity. This diminishes the need for contraction of the antigravity muscles, which have reflexively contracted to splint the inflamed spinal segment. How traction improves the benefit from mere bed rest has not been confirmed.

Distraction of the spinal functional units has been claimed. Some distraction undoubtedly occurs with effect on the disk pressure.<sup>156</sup> Passive traction implies a continuous static force being used for a varying period of time, whereas active traction implies varying forces applied via traction or traction coupled with active exercise. With passive traction the intervertebral disk pressure remained close to the resting pressure, whereas when traction was active, the resultant muscular contraction increased the intervertebral disk pressure, possibly from the concurrent paraspinal muscular activity.

Further studies with autotraction decreased symptoms, but computerized axial tomography (CAT) scanning failed to confirm any change in the disk size or shape.<sup>157</sup> The amount of traction would probably require hundreds of pounds to accomplish significant disk change, which would not be clinically feasible. Physiological studies suggest that the applied traction weight must be 25% of the body weight to overcome the inertia and resistance of the supine body and achieve any distraction of a lumbar functional unit.<sup>158</sup>

Traction decreases lumbar lordosis. The effects of this on the lumbosacral spine are summarized as:

1. Opening the intervertebral foramina.
2. Separating the zygapophysial joints.

3. Elongating the erector spinae muscles.
4. "Stiffening" the annular fibers of the intervertebral disk, thereby unloading the internal pressure within the nucleus.
5. Decreasing the length of the nerve roots and their dura, thereby decreasing the tension on them.
6. Questionably reducing any nuclear bulging within the disk, thereby diminishing bulging or protrusion.<sup>159-163</sup>

**Methods of Application.** There are numerous methods of application of pelvic traction, with the most common being that to the supine patient. A pelvic belt that is molded to the contours of the patient's pelvis is the site of bodily attachment (Fig. 10-55). To this girdle are attached lateral straps that are attached to a spreader bar. A rope from this bar is then attached to gradated weights through a series of pulleys.

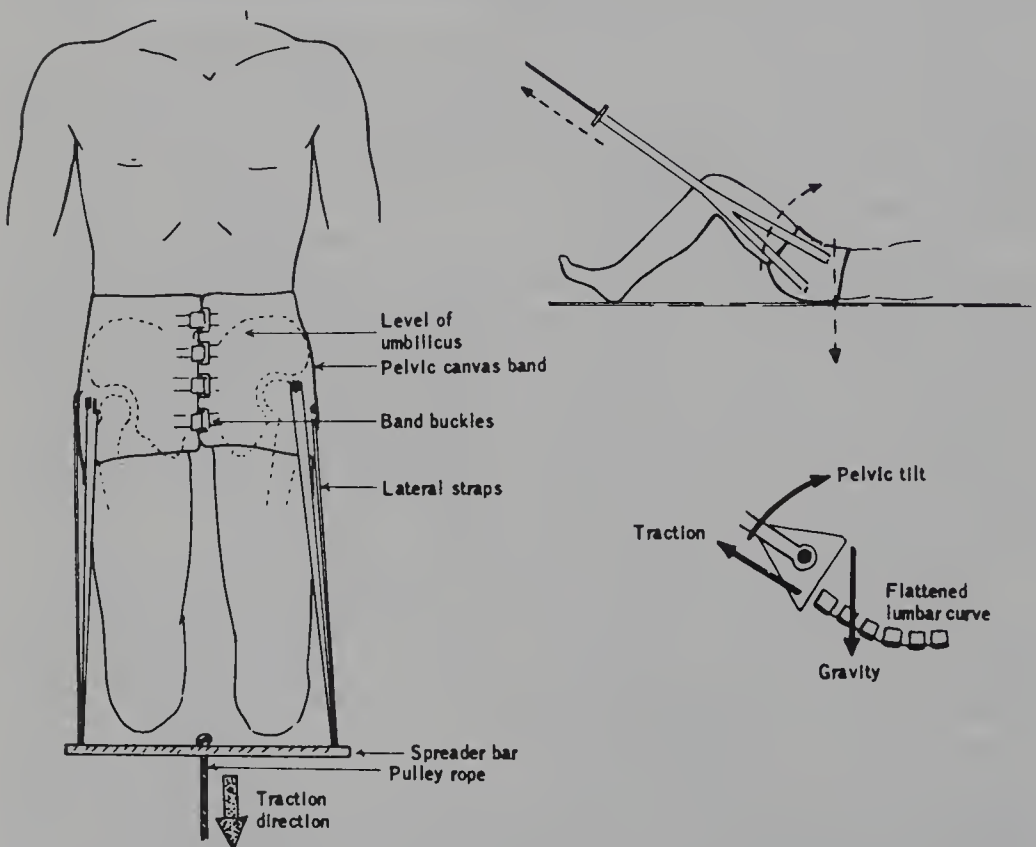


Figure 10-55. Equipment for pelvic traction. Drawing on the left shows the pelvic band placed over the bony pelvis with lateral straps that attach to a spreader bar to the traction equipment. The right drawing shows the angle of pull to rotate the pelvis (arrows) as well as to apply lumbar distraction. The lower left portion of the figure clarifies the intent to flatten the lumbar curve (decrease the lordosis).



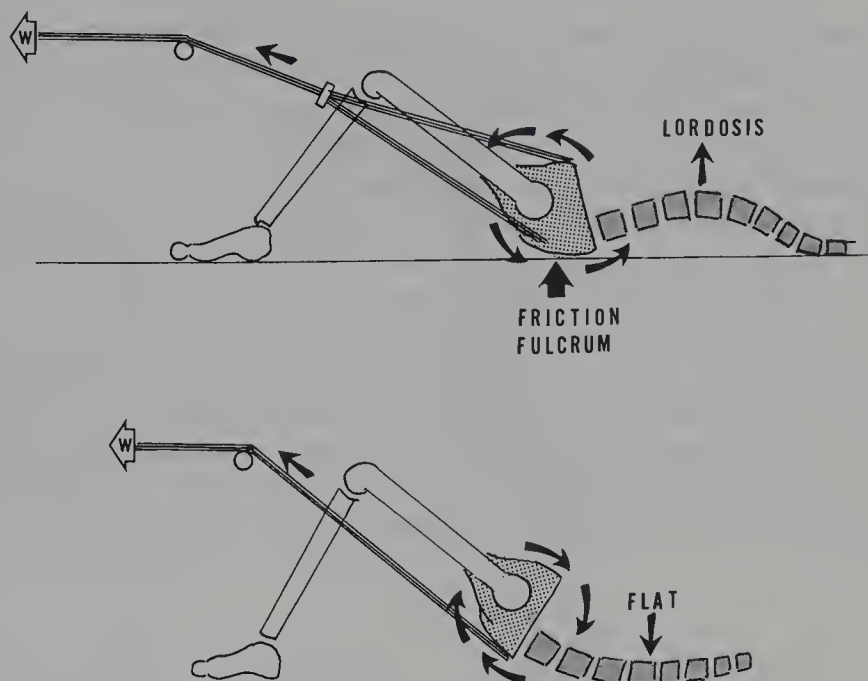


Figure 10-56. Pelvic traction principle. The upper drawing shows distraction (*small straight arrow*) with rotational forces around the pelvis (*curved arrows*). The pelvis remains against the bed or table with friction, causing a fulcrum about which the pelvis rotates.

The angulation of the straps determines the direction of pull on the pelvis (Fig. 10-56). While in the traction, the position of the body implements the purpose of the traction by varying the degree of knee flexion.

Autotraction, meaning a manually exerted force applied through traction equipment (Fig. 10-57), is an active traction, wherein the

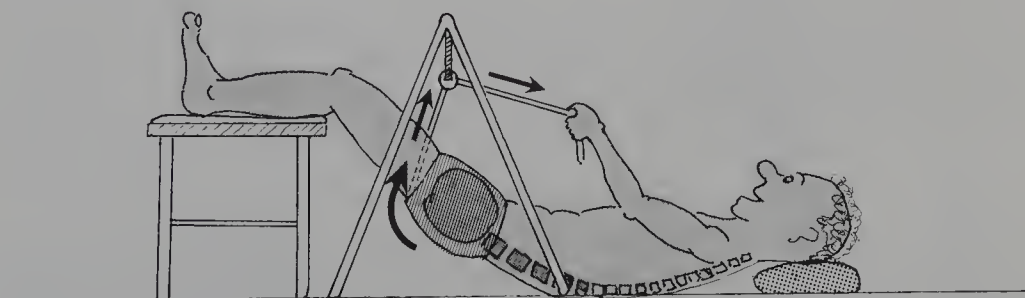


Figure 10-57. Active manually operated home pelvic traction. With the equipment shown, traction can be actively applied at home. The height of the pulley determines the rotational forces and the person determines the amount of manually applied force for distraction.



force and duration are manually controlled by the patient. In spite of active muscular effort by the patient's arms, this traction is actually passive traction. The same principles apply, but it is easier to institute, whereas other forms of pelvic traction need assistance.

Manual-mechanical traction has been advocated, especially by chiropractors, using a divided table to which the patient is strapped. The table divides to apply traction.<sup>164</sup> This requires special equipment and demands transportation of the patient to an office setting.

Autotraction, meaning self-applied traction by the patient, also can be accomplished by inversion traction, where the feet or legs are immobilized and the inverted patient uses the body weight for the traction force. Several types of inversion traction are available. Padded boots with a hook that is held by a chinning bar is one type of inversion traction (Fig. 10-58). On the left side of Figure 10-58 is a similar type

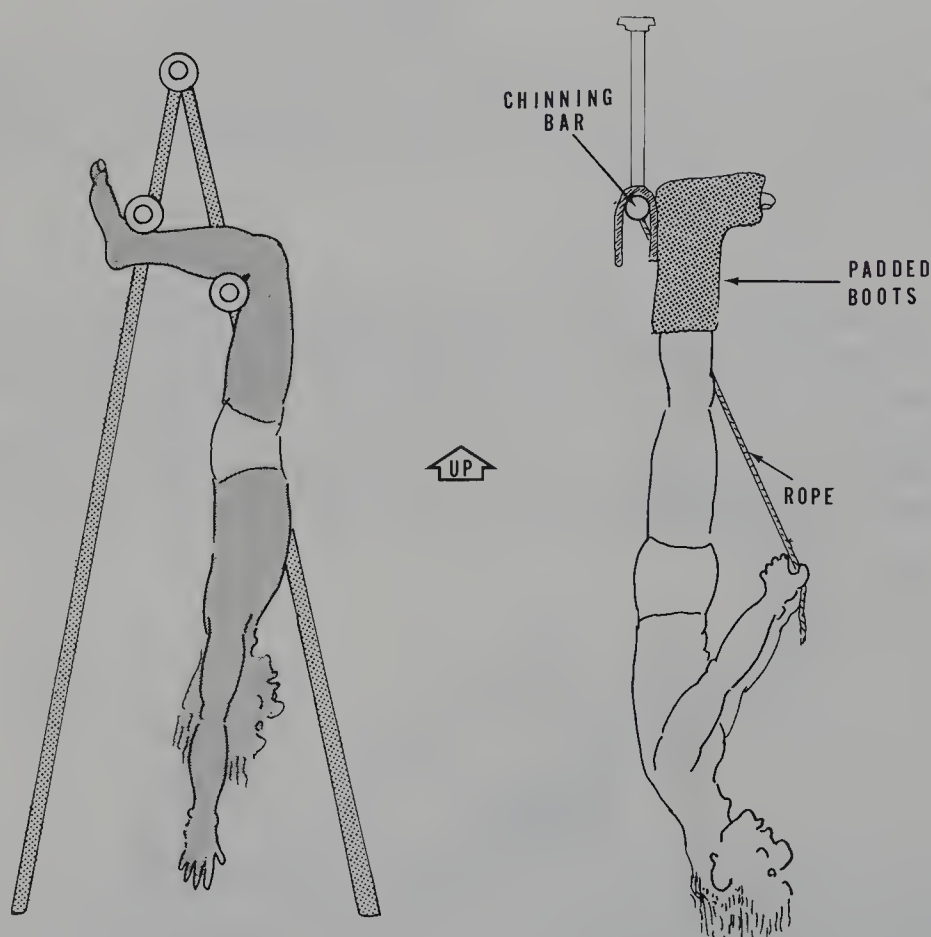


Figure 10-58. Gravity inversion traction. Two manners of applying gravity inversion traction. *Left* shows use of two parallel bars and *right* the use of special boots that hook over a bar.

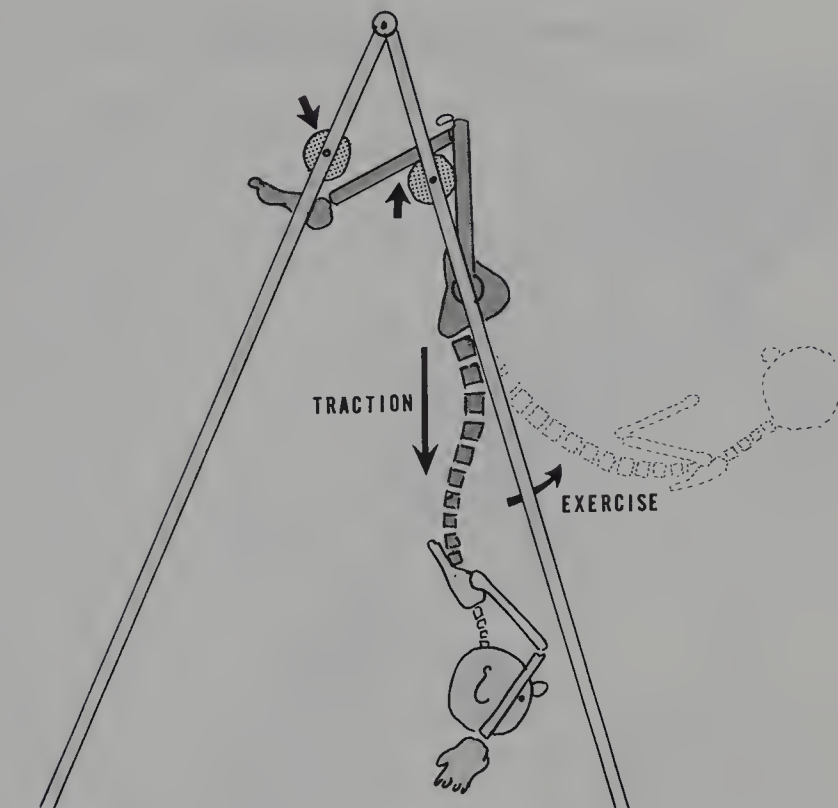


Figure 10–59. Exercise within gravity traction equipment. Within the gravity traction equipment the person can perform flexion or extension exercises.

of inversion traction without boots. While a patient is in this traction, mere passive traction is possible, wherein the body weight is the force. Exercises can be performed while in the dependent traction position (Fig. 10–59).

An elaborate gravity traction is available, wherein the patient is held in a thoracic garment and the straps are brought cephalad to attach overhead (Fig. 10–60). In this apparatus the legs and pelvis act as the traction force and the angle is varied by the rotatory equipment. It has been estimated that 30% of the total body weight is located below the third lumbar vertebra.

Inversion traction, which holds the head down, has been questioned because of the allegation that this position causes increased intraocular pressure, jeopardizing a patient by causing or aggravating glaucoma. Hypertension is also claimed to be adversely affected by the head-down position. Both of these have been refuted,<sup>165</sup> but nevertheless the value of pelvic traction per se has been determined to be ineffectual by the Quebec Task Force on Spinal Disorders.<sup>166</sup>

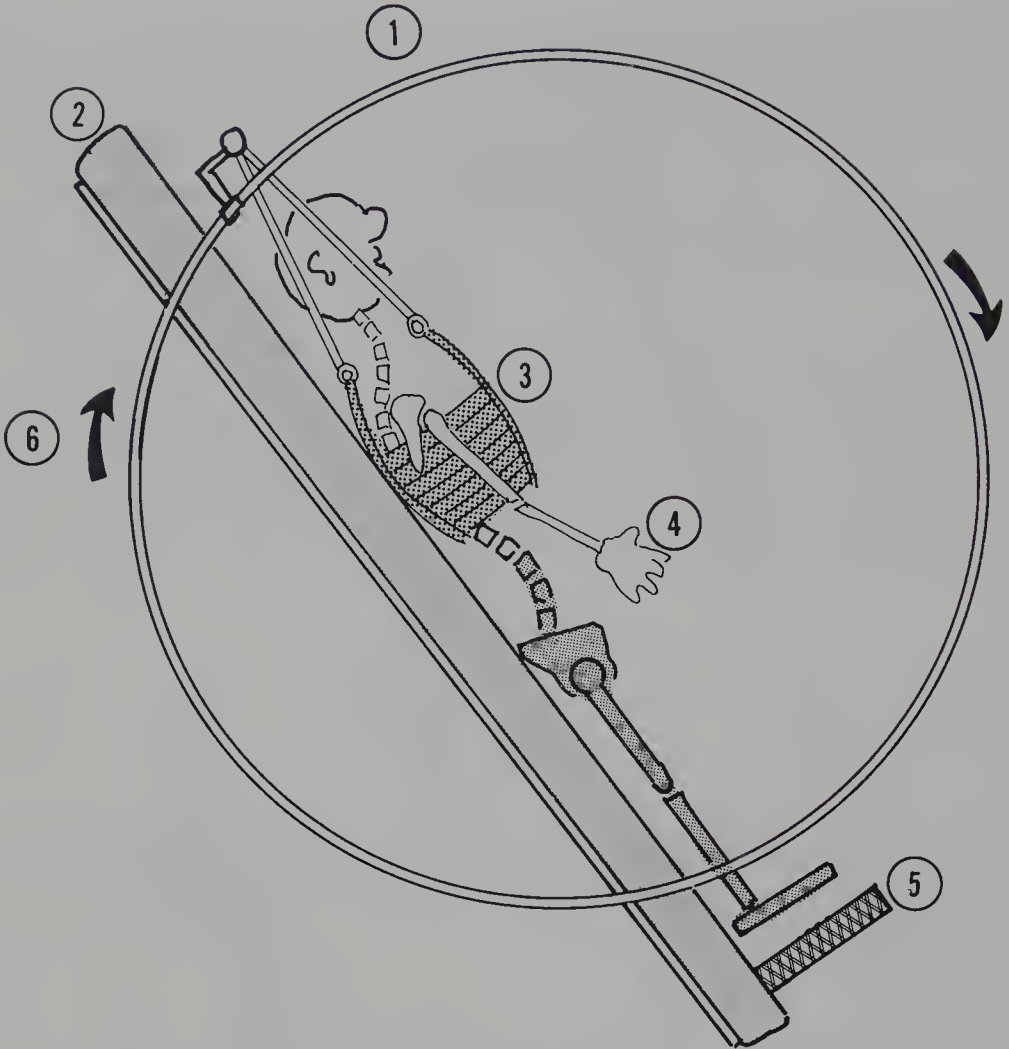


Figure 10-60. Thoracic corset pelvic traction. With a thoracic corset (3) the lower body gives the distraction force. Placed in a circular bed (1 and 2), changing the angle of rotation, the force increases as the vertical approaches (6). The hands are free (4) and a foot plate (5) prevents further slipping.

Research and clinical documentation do not validate using pelvic traction, but if a home program is initiated that causes the patient to claim benefit, it is valid. But repeated episodes of clinic-office traction without objective benefit and change in the organic signs and decrease of documented disability are to be condemned.

## ANCILLARY MODALITIES

In the treatment of acute and chronic pain when the modalities that have been advocated fail to permit the patient to regain function, other modalities have enjoyed acceptance.

## Transcutaneous Electrical Nerve Stimulation

For many years, transcutaneous electrical nerve stimulation (TENS) has been advocated as a treatment for acute and chronic pain,<sup>167,168</sup> including low back pain.<sup>169–174</sup> Its efficacy has been questioned in more recent studies.<sup>175</sup> As its efficacy has been asserted exclusively for subjective pain and for the functional limitations imposed by pain, it cannot be evaluated objectively.

In the physical treatment of pain, TENS has been well accepted as being effective. Its neurophysiological basis has also been established.

Pain mediation through the C unmyelinated and A alpha lightly myelinated fibers has been established.<sup>176</sup> Large-diameter, myelinated fibers transmit mechanoreceptor impulses and have a lower threshold at their synapse at the dorsal horn. As they transmit at a faster speed than those of the unmyelinated or lighter myelinated, they arrive at the “gate” earlier. The gate concept implies that these impulses block subsequent pain-producing impulses.

Transcutaneous electrical nerve stimulation of low frequency and high intensity of less than 10 Hz has been clinically shown to create analgesia.<sup>176</sup> Endorphins have been created in the absence of pain,<sup>177</sup> and the effect of TENS has been eliminated by simultaneous use of naloxone, which is an indication of a neurochemical basis.<sup>178</sup>

Questions have been raised as to whether there is an increase in endorphins from TENS and what is the appropriate frequency of the TENS to elevate this opium peptide in the cerebrospinal fluid. It was determined that low-frequency (2 Hz) stimulation caused an elevation of Met-enkephalin-Arg-Phe-Dynorphin: an opioid peptide in the cerebrospinal fluid.<sup>179</sup> Higher frequency (100 Hz) caused a lesser elevation of a different opioid. This accentuates the need to determine which frequency is the most effective and which opioid is liberated and indicates that there is an elevation of opioid when the TENS application is effective.

Electrical stimulation has been shown to increase levels of dopamine, epinephrine, and serotonin, which are established algogens.<sup>180</sup> Electrical stimulation has been shown to decrease nerve action potential of A delta fibers,<sup>181</sup> which are the pain mediators. All these factors confirm a physiological basis for effective pain modulation.

The manner, site, and type of TENS are vital for effective pain modulation. TENS is most effectively applied proximally in nerve injuries and the precise site of application must be clinically ascertained. The current wavelength must be determined (Fig. 10–61) as well as its form (Fig. 10–62). A recent report advocated greater relief from the use of ultralow frequency TENS (0.66 Hz) in contradiction to the currently advocated strengths.<sup>182</sup>

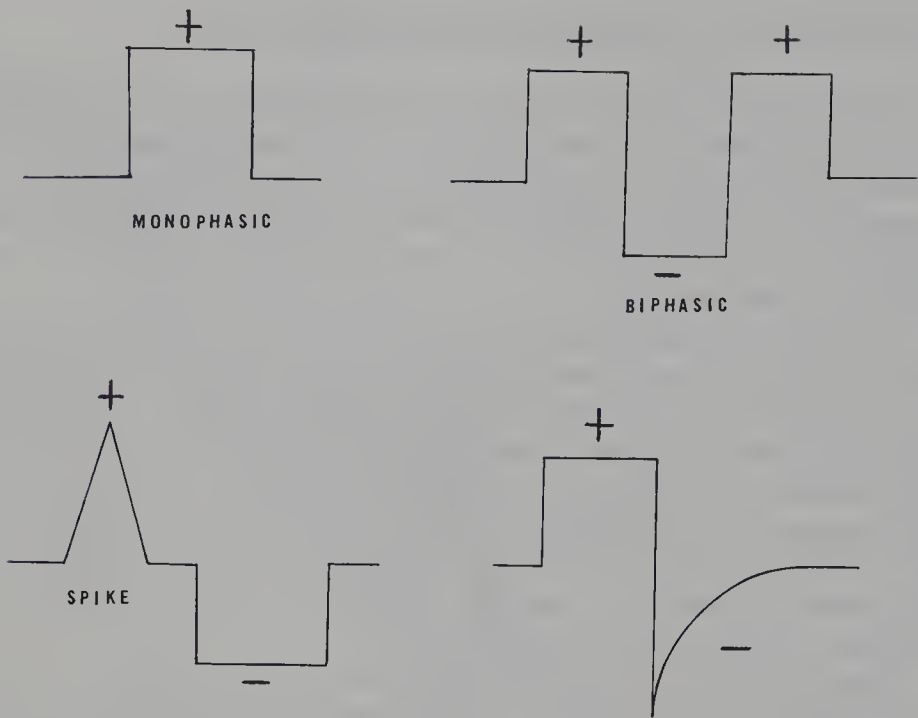


Figure 10-61. Wave forms of transcutaneous nerve stimulation (TENS). Wave forms of TENS.

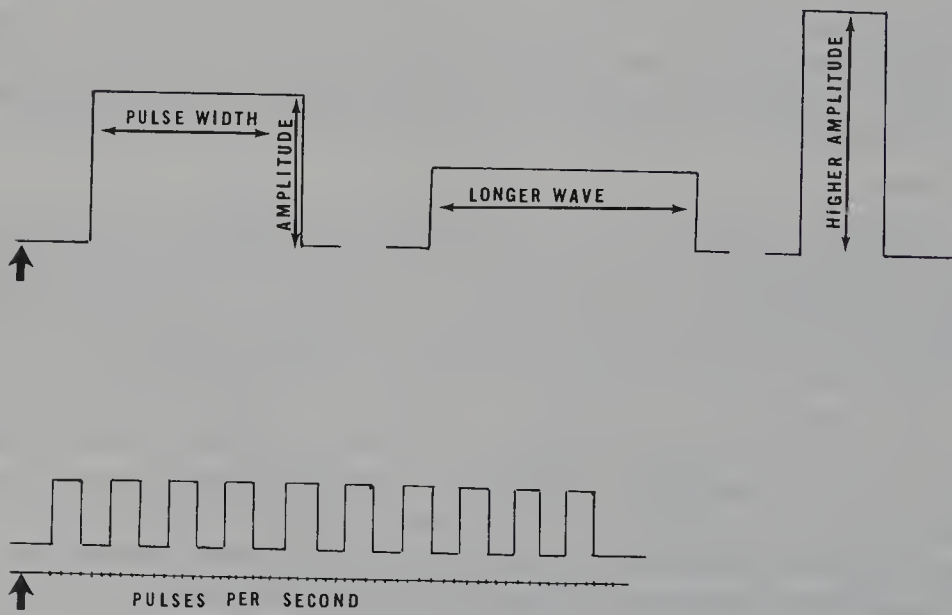


Figure 10-62. Characteristics of TENS application.



The efficacy of TENS in treating chronic pain has varied from 12% to 60%, depending on the reporter.<sup>183</sup> Patients with significant depressive illness complicating their pain receive significantly less benefit from the use of TENS, so TENS cannot be considered to effect pain modulation through its psychotherapeutic benefit.

## Acupuncture

Derived from ancient Oriental medicine, acupuncture has evolved in current Occidental medicine for the treatment or prevention of pain. The modality of acupuncture treatment<sup>184</sup> is essentially the insertion of small (thin) solid needles into the skin, immediate subcutaneous, and muscular tissues in regions considered *meridians*.

Chinese medicine<sup>185</sup> considered human health as the result of conflicting forces of nature termed *yin* and *yang*. When these forces were out of harmony *dis-ease*<sup>186</sup> resulted. There existed a concept of vascular and neurological energy flow through channels termed meridians. These meridians allegedly followed a circadian rhythm,<sup>187</sup> and each was directly associated with an organ system.

The meridians were interconnected within this vital life energy *chi*, and allegedly a deficiency of chi was considered to cause pain. Pain could thus be modified by inserting needles into the meridians that rebalanced energy flows. More recent concepts associate the meridian process with the autonomic nervous system.<sup>188,189</sup>

There are numerous techniques for inserting acupuncture needles that have not been officially recognized. The angle and depth of the insertion vary, and numerous techniques exist, such as twirling, or twisting the inserted needle; pressing down on the needle; and applying a ball of herbs at the base and igniting the ball.

The site of insertion appears to be the major basis for success. The sites of trigger points or motor points<sup>190</sup> have been postulated as the optimum sites for acupuncture.

The physiological basis for acupuncture remains unconfirmed.<sup>191</sup> Its efficacy and validity as an accepted medical modality remain ambiguous by Western medicine. In 1981 the American Medical Association decided that there was insufficient evidence to conclude that acupuncture had no more effect on pain than placebo or sham acupuncture.<sup>192</sup>

Animal studies are themselves inconclusive, because stress is imparted to the animal during the acupuncture experiment and stress itself is analgesic.<sup>193</sup> The results of animal studies cannot be translated to humans. Human studies are also nonspecific, although there has been evidence that endorphins are elevated in the plasma,<sup>194</sup> but their transfer of these endorphins to the central nervous system remains unstudied.

Clinical studies of acupuncture are subjective, as most pain studies are expected to be. The mere belief that acupuncture can be effective influences the patient reaction as to the relief of pain from an application.<sup>195</sup> Studies have revealed that acupuncture is no more effective than TENS at the same tissue sites.<sup>196</sup> Cultural background as an influence on the benefit from acupuncture has been studied and found not to be a significant factor.

## Epidural Analgesia

Epidural analgesia, often with steroids, has been used for many years in treatment of pain and especially in chronic low back pain or low back pain with radicular pain that does not respond to the usual conservative managements.<sup>197</sup> A narcotic or analgesic produces analgesia by two mechanisms. First, a portion of the drug crosses the dura mater and enters the cerebrospinal fluid, where it penetrates into the dorsal horn of the spinal cord. Second, there is systemic absorption as occurs from intramuscular injection.<sup>198</sup>

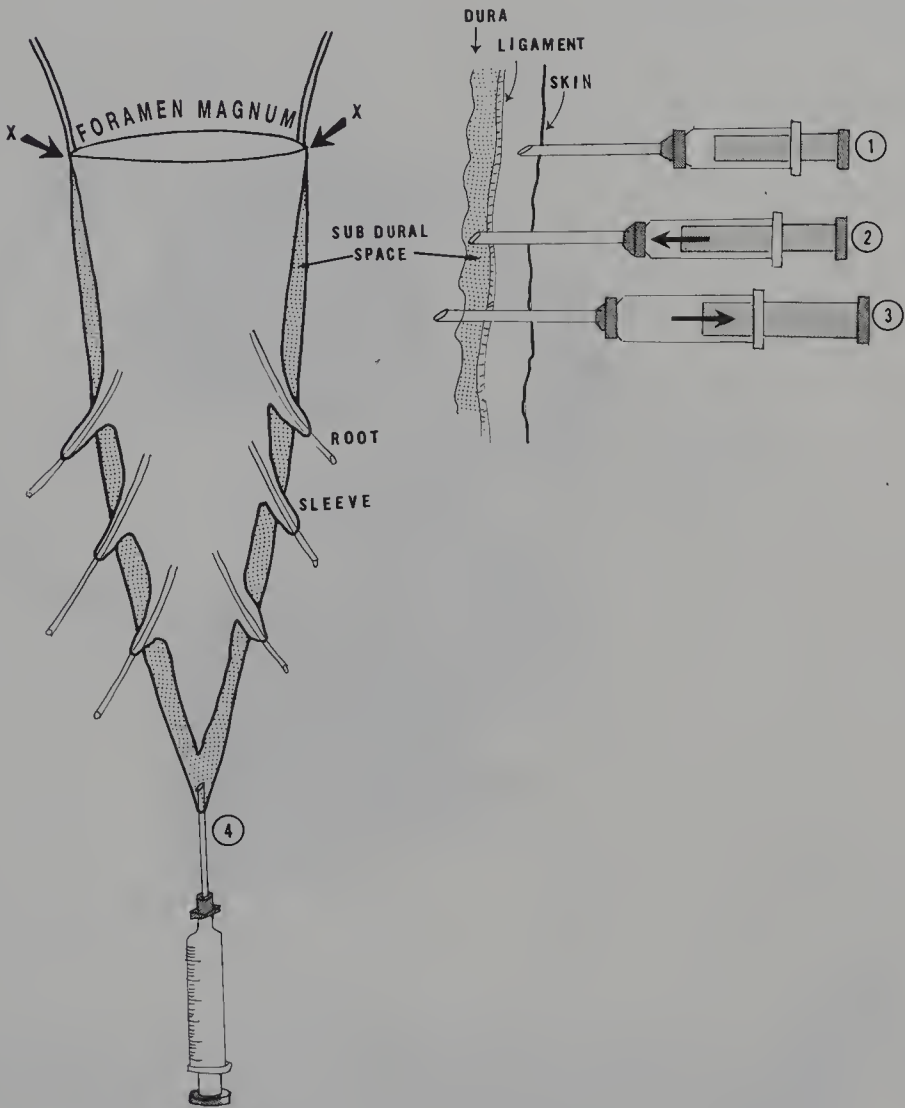
There is a negative pressure within the epidural space that facilitates the injection route and ensures acceptance of the analgesic agent and steroids into that space.

The injection varies from the caudal route or entry at the lumbar level. The former is often ineffective as the caudal canal is obliterated in many people, whereas the lumbar site is usually patent.

The technique is the insertion of the 20-gauge short beveled spinal needle attached to a syringe that is only partially plunged. As the dura is approached through the thick ligamentum flavum and penetrated, the plunger of the syringe is depressed (into the barrel), whereas if the penetration proceeds further *into* the dural space, the spinal fluid has a positive pressure that ejects the plunger and fills the syringe (Fig. 10-63). Once the epidural space is entered, the injectable fluid is entered. The basis of the epidural route as compared with intradural or peripheral nerve block is illustrated in Figure 10-64. The injected analgesic and steroid act on the afferent neurological pathways (Fig. 10-65), affecting the low back and sciatic radiculopathy.<sup>59,199-201</sup>

If it is the intention to insert a catheter for prolonged injection of analgesic or steroid, an 18-gauge thin walled needle that allows passage of the catheter is used<sup>202</sup> (Fig. 10-66).

Nerve root injections have also been used for diagnostic attempts to determine the exact nerve root level in radiculopathy syndromes. Steroids injected into the paraneural area of the nerve root (Fig. 10-67) or into the dorsal root ganglion also have their advocates (Fig. 10-68).



**Figure 10-63.** Technique of epidural injections. The dura is attached to the bony canal at the foramen magnum (X), following which a subdural space is present. On the right (1) the spinal needle penetrates the skin and the plunger does not move. (2) As the needle penetrates the ligament into the subdural space, due to a negative pressure, the plunger is pulled into the syringe. (3) If the needle enters the dural space, the spinal fluid exerts positive pressure then rejects the plunger from the syringe and fluid enters the syringe. (4) Site of needle entrance into epidural sac for caudal injection.

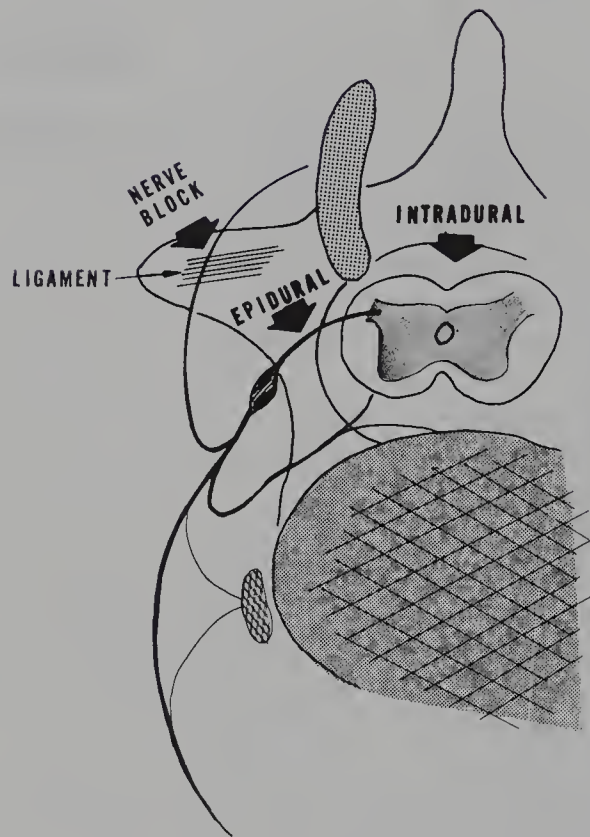


Figure 10-64. Epidural site of injection compared with intradural site of injection or nerve block. The intradural site approaches pain from dural anti-inflammatory medication. The epidural site blocks primarily autonomic fibers. Nerve block interrupts the posterior primary division of a peripheral nerve root.

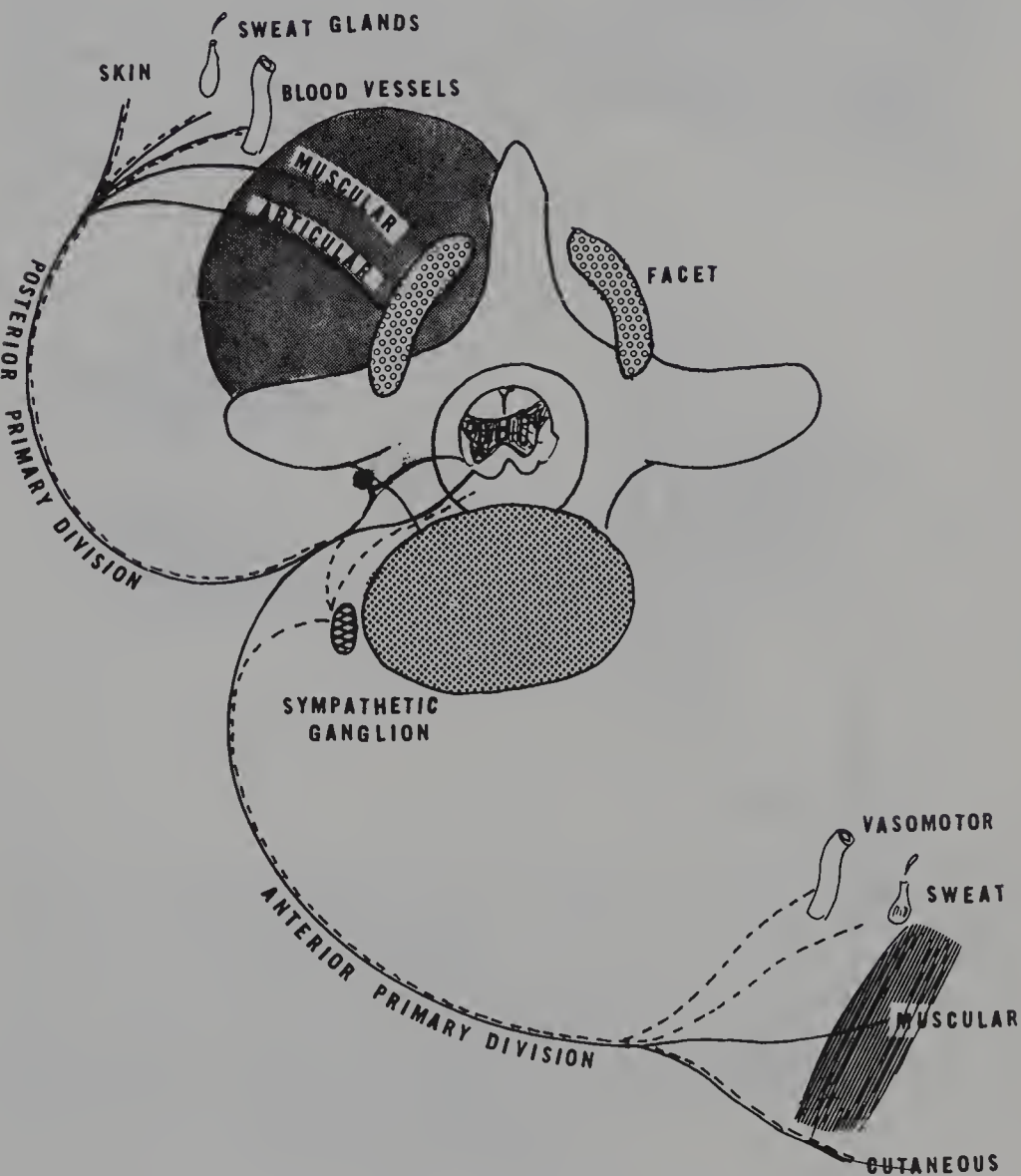


Figure 10-65. Neurologic pain pathways. The neurologic pathways by which nociceptive impulses are sent via afferent fibers to the cord. The autonomic motor pathways are also depicted.



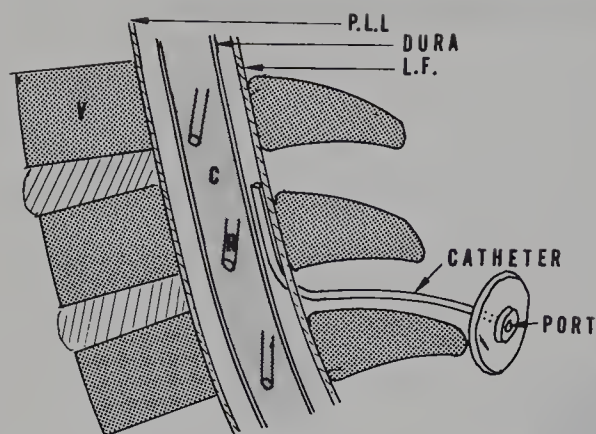


Figure 10-66. Epidural anaesthesia by indwelling catheter. If prolonged infusion of epidural anesthesia or anti-inflammatory medication is indicated the technique is to insert a catheter into the epidural space where it remains during the treatment. PLL is the posterior longitudinal ligament, and LF is the ligamentum flavum (noted in the cervical spine). The port is the site through which the medication is injected.

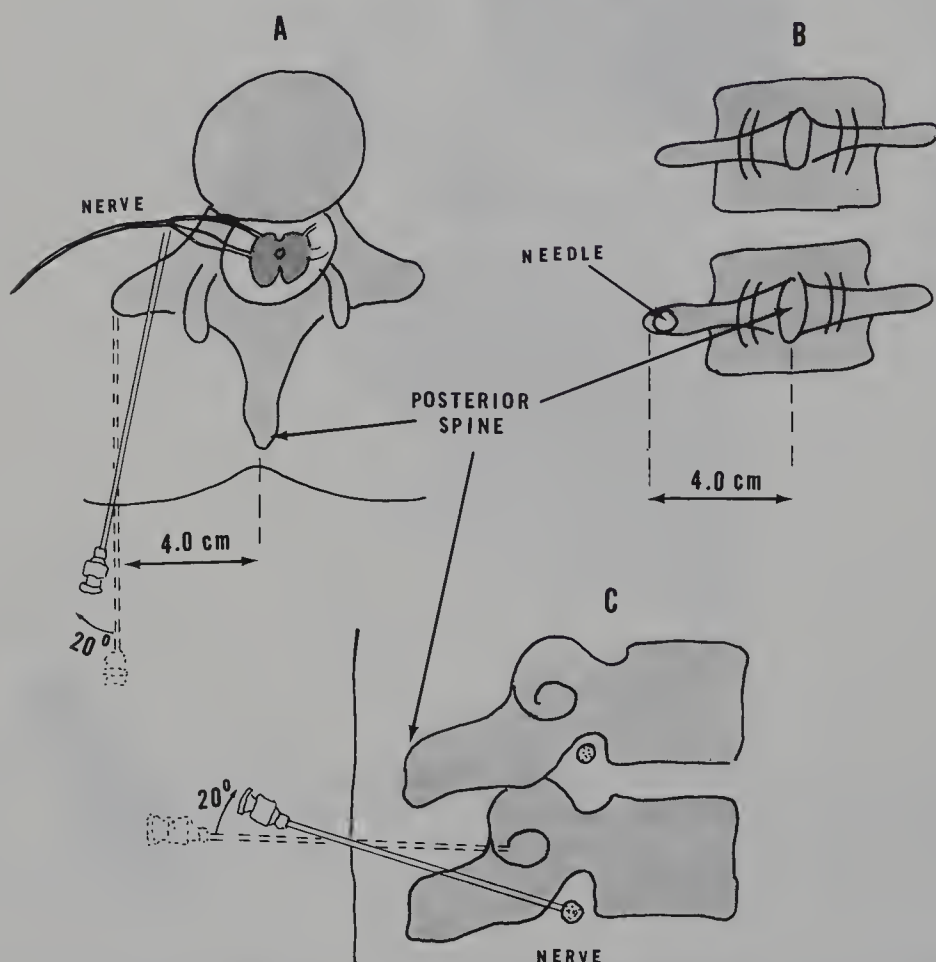
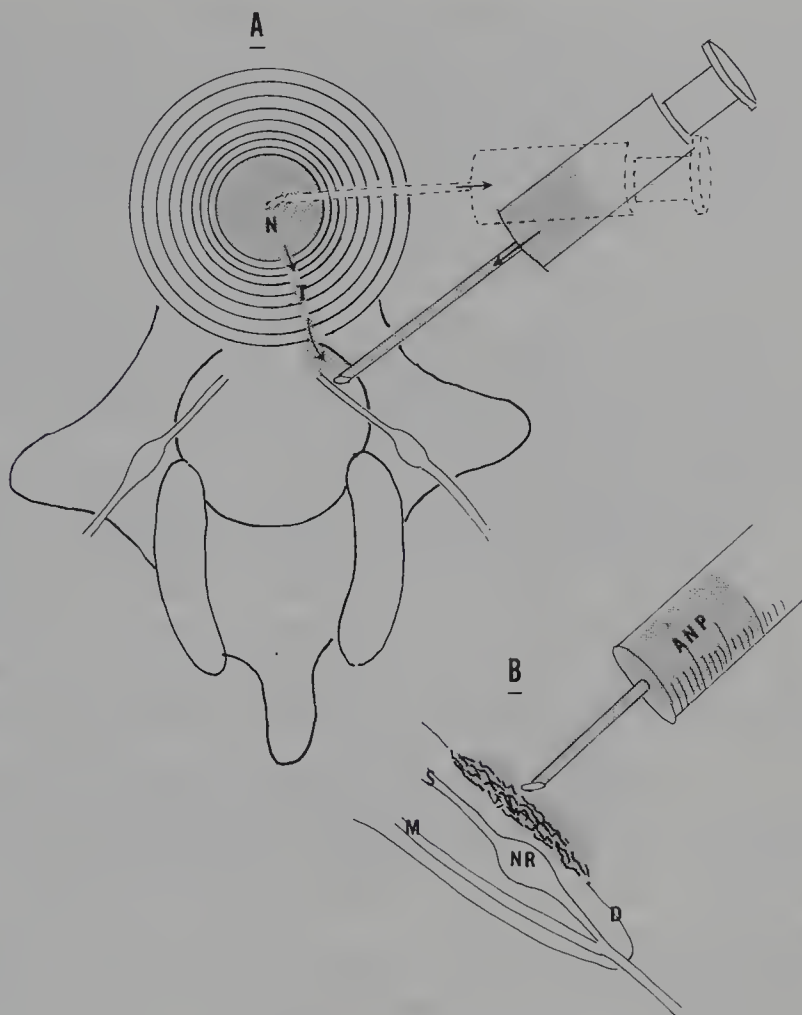


Figure 10-67. Technique of nerve root injection. With the patient in the prone position and a pillow under the abdomen an 8 to 10 cm 21-gauge needle is inserted 4 cm lateral to the upper margin (A) of the spinous process until it penetrates to the lower margin of the same vertebral transverse process (B). Upon contact with the process the needle is slightly withdrawn and directed medially (A) and 20 degrees downward (C). This directs the needle towards the nerve root (C) which, when contacted, elicits pain in the dermatomal area.



**Figure 10-68.** Injection of analgesic and steroid into the area of nerve root and dorsal root ganglion. When an extruded disk (A) encroaches on a nerve root through a tear in the annulus (T) the extruded material entraps the root. Material of the annulus (*dotted syringe*) can be aspirated from the remaining disk nucleus and injected into the site of the extruded material (B). This is near the sensory (S) and motor (M) roots as well as the dorsal root of the nerve (NR). The needle penetrates the dura (D) in this injection. An analgesic and steroid can be injected into this site rather than nuclear material.

Facet injection of an analgesic or steroid to relieve pain asserted to be emanating from the facets has been discussed in Chapter 6.

Injections into the epidural space and injection of the nerve roots should only be considered when noninvasive modalities have failed and there are organic signs found causing the impairment leading to disability.

## Pharmacological Treatment of Low Back Pain

The most commonly used drugs in the treatment of low back pain include pure analgesics, nonsteroidal anti-inflammatory drugs, steroids, muscle relaxants, and antidepressants. Narcotic analgesics are used only when there is severe pain not responding to the lesser analgesic medications.

Analgesics are agents that are considered to relieve pain by acting centrally. A possible mechanism has been postulated by the discovery of opiate receptors<sup>203</sup> in selected areas of the central nervous system and of endogenous substances termed *enkephalins* (*endorphins*). The receptor sites have been termed *mu*, *delta*, and *kappa*.<sup>204</sup>

Nonsteroidal anti-inflammatory drugs are numerous and must be used with concern because of their potential undesirable side effects, especially gastrointestinal reactions. Most of these drugs have not proved to exceed the benefit from aspirin.<sup>205</sup> Muscle relaxants have been nonspecific, as most drugs in this category are also sedative or antianxiety drugs. The drugs used for treating neurological spasticity (such as baclofen) have been used, but the literature varies as to their efficacy.<sup>206</sup>

Studies have suggested that carisoprodol is superior to alternative medications such as butabarbital, diazepam, or a placebo.<sup>207</sup> Diazepam was found not to be more effective than a placebo.<sup>208</sup> Sedatives and antianxiety drugs, therefore, have not been proved more effective than other medications and modalities.

Antidepressants have been increasingly used in the treatment of patients with acute as well as chronic low back pain not only for their action against the prevalent depression that accompanies the usual low back pain but also as they have proved to be chemically effective in pain management.

Opiate receptors are located in the medial thalamus as well as in areas of the cord gray matter, dorsal columns, and the Rexed layers I and II. The presence of these receptors indicates that the body is capable of manufacturing its own narcoticlike substance. It is hypothesized that opioids inserted at these sites activate the descending pathways to the midbrain periaqueductal gray matter,<sup>209</sup> which inhibits ascension of nociception from the cord level.<sup>210</sup>

A recent study has postulated that opioids have their effects at the peripheral mechanism as well.<sup>211</sup> This finding was in patients undergoing arthroscopic knee surgery, and the mechanism remains obscure but provocative.

Opioids, as well as acting upon the *mu*, *alpha*, and *kappa* receptors, also act upon the postganglionic sympathetic terminals that block release of prostaglandins, which are thought to be involved in sympathetic maintained pain syndromes.

Opiates are markedly underused in treating pain, causing patients to suffer needlessly.<sup>212</sup> This is partly due to the myth of opiate tolerance and addiction.<sup>213</sup> The problem has been compounded by restrictive laws regarding opiates, with attendant social, political, and legal problems. This is not to advocate the indiscriminate use of opioids but to state that brief, carefully moderated use of opioids in patients suffering severe acute low back pain may lead to earlier recovery and resumption of beneficial activity.

The use of opioids in the treatment of chronic low back pain is more complicated. For many years, the long-term use of opioids for chronic pain was forbidden or at least contraindicated despite the fact that their use was highly effective. Sternbach<sup>214</sup> questioned the safety and “humaneness” of opioids in the light of a 5% iatrogenic rate and minimal functional improvement that accompanied opioid therapy.

There are guidelines that have been postulated for long-term use of opioids<sup>215</sup>:

1. Persistent pain is a major impediment to recovery of function.
2. Psychological problems are not present or considered pertinent.
3. All other nonnarcotic analgesics have failed.
4. There is no prior history of substance abuse.
5. A primary physician assumes responsibility for monitoring the medication and its response.

The difficulties in implementing these guidelines in acute low back pain are:

1. There are no objective criteria that pain is the sole or major impediment to function.
2. It is hard or even impossible to ascertain the absence of psychological factors or the degree to which the psychopathology is the result of the pain.
3. It is usually unclear whether the depression is a factor in the pain and its resultant dysfunction and whether opioids will enhance or decrease this depression.
4. The previous use of nonnarcotic analgesics has been appropriate, with a sufficient duration and dosage.
5. Compliance is ascertained or ascertainable.

A dictum must be applied that medications be appropriately administered.

Medications have a positive reinforcement of the “benefits” of pain and thus must be monitored, keeping in mind that the prescription and ingestion of drugs may cause a euphoria and an unrealistic loss of pain not requiring other intervention activities by the patient.

The use of “p.r.n.,” meaning the ingestion or injection of a pain medication “upon request,” must be avoided as it focuses the impression of pain and its severity on the patient, the practitioner, and often the family. If medications for analgesic purpose are prescribed, they should be given “q. 6 hours” or “q. 4 hours” and for a specific duration

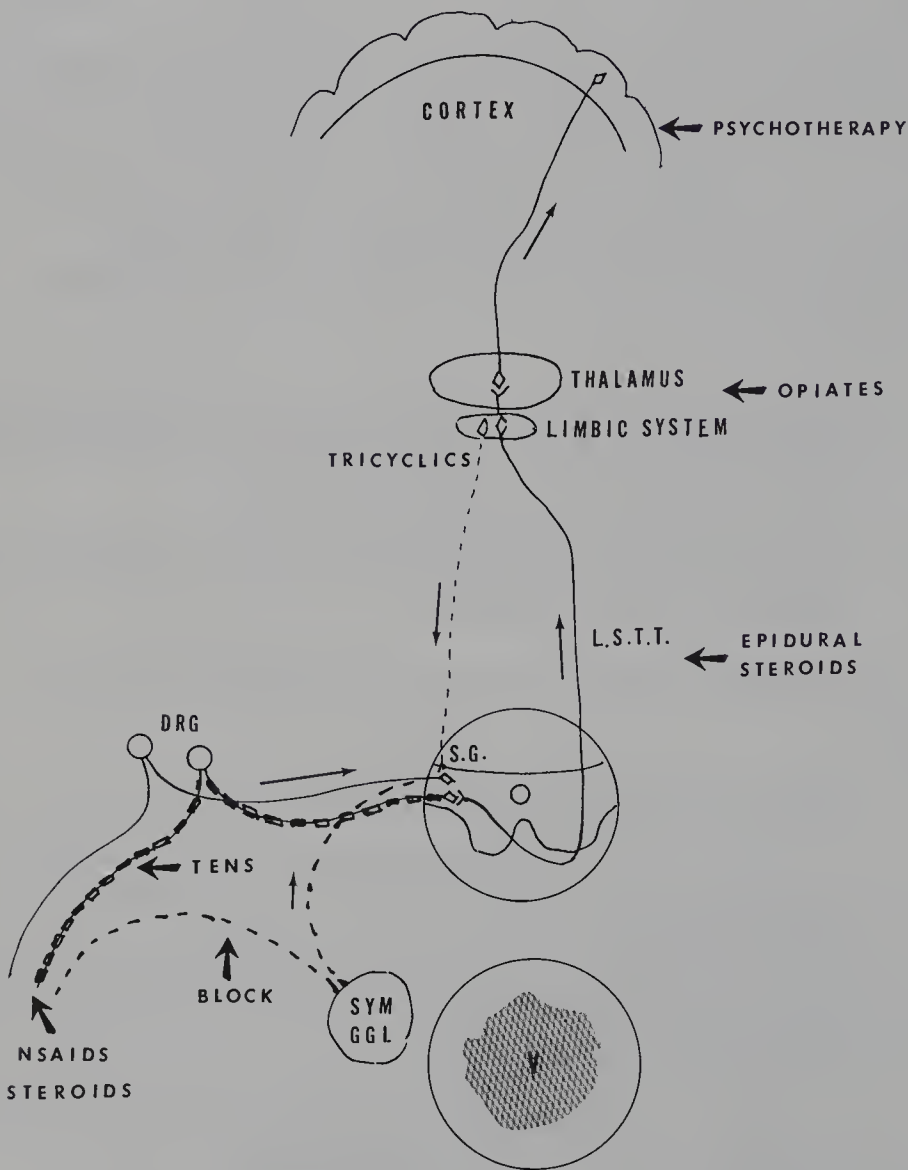


Figure 10-69. Sites of action of medications in the treatment of pain. Nonsteroidal anti-inflammatory drugs (NSAID) and steroids act on the peripheral nerve and dorsal root ganglia (DRG). TENS blocks impulses transmitted through afferent fibers and anesthetic nerve blocks interrupt the autonomic fibers. All are blocked before entering the substantia gelatinosa (SG). Epidural steroids interrupt the lateral spinothalamic tracts and tricyclic drugs intervene at the limbic area where opiates also act. Psychotherapy acts at the cortical level.



of time and amount with specified decrease in frequency and duration. This denotes that the benefit will be derived from a precise period of time and a specific dosage of medication. With this type of administration of medication, pain is no longer the criterion for which medication is prescribed and function becomes the desirable end point of treatment.

Current concepts of treating chronic pain mandate that the initial intent be to decrease or eliminate the dependency of the patient upon pain medications, especially if they are addictive or become less effective from prolonged use. If dependency on drugs is initially avoided, their later elimination in treating chronic pain results.

A series of chemical reactions in nociception occurs within the central nervous system that once established may perpetuate and increase. This indicates the need for early intervention based on a full understanding of the mechanisms of pain production and transmission (Fig. 10-69).

There is a neuroplasticity within the pathways of nociceptive impulses from the end organs, through the dorsal root ganglia, the dorsal horn neurons, and the ascending cord pathways that increases sensitivity and furthers the transmission of the chemical reactions that ultimately result in subjective pain.<sup>216</sup>

The resultant spasm that accompanies pain from inflammation of tissue sites in low back pain is attributed to a vicious cycle maintained by a pain-spasm-pain feedback. This feedback loop has been postulated but as yet not confirmed.

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## CHAPTER 11

# Miscellaneous Lumbosacral Spine Conditions Causing Pain and Impairment

There are numerous other conditions of the lumbosacral spine, congenital or acquired, that may be directly related to causing “mechanical” low back pathology.

### SPONDYLOLYSIS AND SPONDYLOLISTHESIS

Herbiniaux,<sup>1</sup> a Belgian obstetrician, first drew attention to the spondylolisthesis lesion when it was considered as an obstruction during labor. Kilian<sup>2</sup> pointed out that the lesion was due to a slow displacement of the last lumbar vertebra and coined the term *spondylolisthesis*. Robert,<sup>3</sup> by dissecting all the soft tissues around the fifth lumbar vertebra, demonstrated that there was no more slippage if the neural arch was intact. It was Hartmann<sup>4</sup> who noticed that although the vertebra slipped forward, the spinous process remained in place, indicating a division in the neural arch. Neugebauer<sup>5</sup> discovered the lack of continuity in the pars interarticularis (lysis). Newman<sup>6</sup> wrote his classic paper on the etiology of spondylolisthesis, and it remains the basis of understanding of the condition.

These conditions are frequently related yet may exist separately. The term *spondylo* relates to the spine, and *lysis* means dissolution. *Listhesis* is from the Greek word “olisthesis,” meaning slipping or falling.



*Spondylolysis* indicates an anatomic defect that causes discontinuity in the pars interarticularis. The proximal segment resides within the lamina. The defect may be unilateral or bilateral, and the widening varies as to degree. There are numerous theories as to etiology, differing from a congenital defect to an intra-uterine or early postpartum fracture. This lesion is often found incidentally in radiological studies, with no clinical significance. It remains considered a weakness in the neural arch that predisposes to ultimate listhesis.

*Spondylolisthesis* indicates a forward or backward translation subluxation of the body of the superior vertebra upon its immediate adjacent inferior vertebra within a functional unit (Fig. 11-1). The common usage usually implies forward slipping of the L5 vertebra on the sacrum

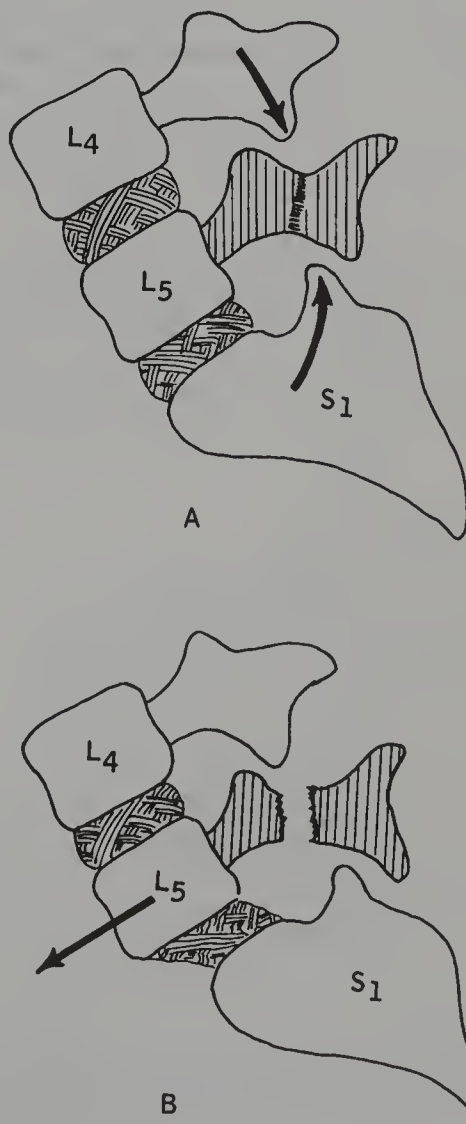


Figure 11-1. Mechanism of spondylolisthesis. (A) The defect in the pars interarticularis (*shaded area*) is depicted but there is no listhesis. (B) There occurs a fracture with separation of the pars and forward listhesis of L5 (*straight arrow*). The *curved arrows* in panel A are considered to be the basis of fracture from severe extension (*curved arrows*).

but may also be forward slipping of L4 on L5. Statistically, 70% of reported spondylolisthesis cases occur at the former, with 25% at the latter and only 4% above the fourth lumbar segment.<sup>7-11</sup>

Normal forward shear of a superior vertebra on its immediate inferior vertebra is considered to occur from the restriction of the facets. The translation that occurs during spinal flexion is arrested by contact of the facets<sup>12</sup> (Figs. 11-2 and 11-3). Forward shear is also limited by the capsules of the facets, the posterior ligaments, and the integrity of the annular fibers of the disk, but the integrity of the facet relationship is predominant.

When there is a defect in the isthmus, mechanical listhesis is permitted. These defects have been classified and quantified.<sup>13,14</sup>

*Type I (isthmic).* There is an anatomic defect in the pars interarticularis (Fig. 11-1). It is seen usually in adolescents and considered to be the result of trauma, causing a fatigue fracture. The fracture usually heals with fibrous tissue and becomes stable.<sup>15</sup>

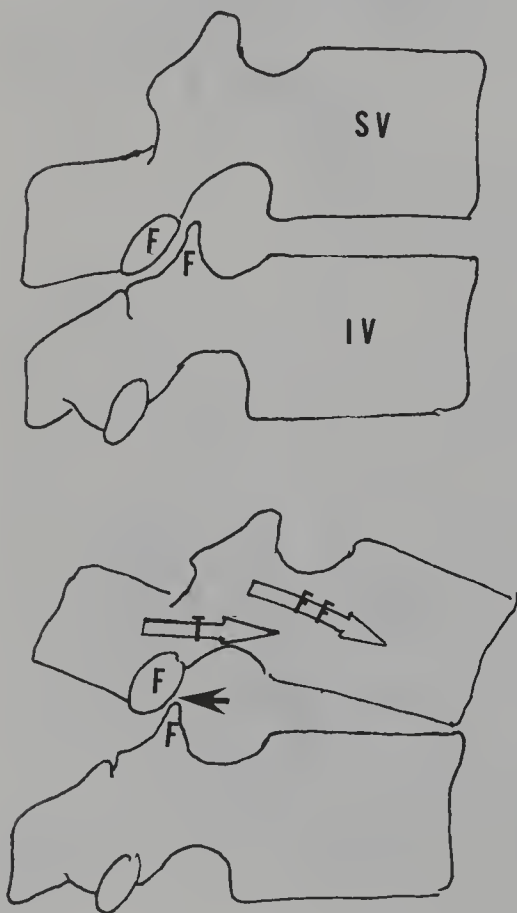
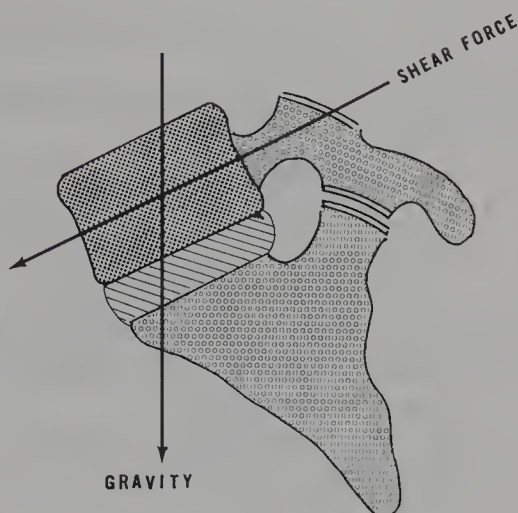


Figure 11-2. Translation flexion forces on the facets. The *upper figure* demonstrates the neutral spine with the superior vertebra (SV) on the inferior vertebra (IV). The facets (F) are depicted. In the *lower figure* as the spine flexes forward (curved FF arrow) it also translates (straight T arrow). The facets (F) impinge (dark arrow) preventing further translation. Further flexion is also limited.

Figure 11-3. Gravitational forces acting on the lumbosacral joint. The fifth lumbar vertebra (not numbered in illustration) slides forward causing a shear force. The angulation of the facets posteriorly prevent further slipping. These are reinforced by the ligaments, joint capsules, and disk annular fibers.



*Type II (congenital).* The posterior elements are structurally inadequate due to developmental causes.

*Type III (degenerative).* The facets and their ligamentous supporting structures have become deficient from numerous causes (Fig. 11-4).

*Type IV (elongated pedicles).* The neural arch is elongated, placing the facets more posteriorly. This is essentially an isthmic variant.

*Type V (destructive disease).* This is a secondary manifestation of a metabolic, metastatic, or infectious disease. Systemic bone pathology can be involved.

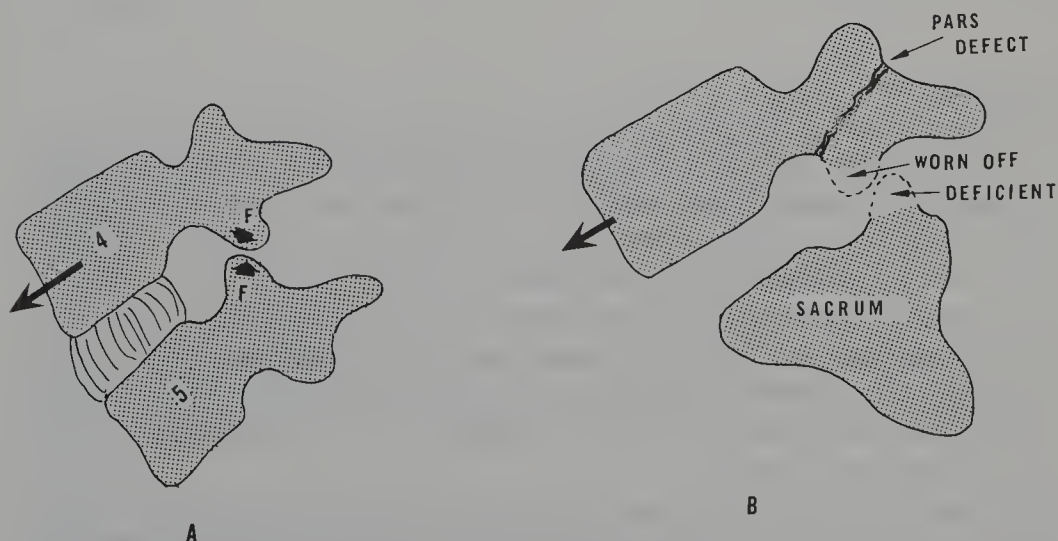


Figure 11-4. Type III spondylolisthesis. (A) The facets (F) preventing L4 from sliding forward on L5 are depicted. (B) The eburnated facets (worn off deficient) allow the upper vertebra to slide forward in spite of a united lysis.

Wiltse et al.<sup>14</sup> classified spondylolisthesis as:

- I. *Dysplastic*. Congenital abnormality of the upper sacrum or arch of L5
- II. *Isthmic*. Lesion of the pars interarticularis
  - A. Lytic fatigue
  - B. Elongated but intact pars
  - C. Acute fracture
- III. *Degenerative*. Progressive intersegmental instability
- IV. *Traumatic*. Fracture or dislocation of the facet joint, allowing forward displacement
- V. *Pathological*. Loss of stability secondary to pathological destruction of facets or pars interarticularis

The classification is redundant but it does not matter as long as the pathology is recognized and so termed.

Spondylolisthesis type I at L5-S1 spinal space usually does not progress after age 20 years until age 70 years but listhesis above has a higher incidence of neurological signs and tends to progress and may often lead to spinal stenosis.

As was stated, lysis is usually not symptomatic and listhesis may exist throughout a person's life without symptoms, but when symptoms do occur, its presence must be confirmed as being the cause. As Laros writes, "Isthmic spondylolysis and spondylolisthesis are x-rays' diagnosis and do not, by themselves, identify clinical causes of symptoms. . . . As such, these x-ray findings do not provide a definitive guide to treatment."<sup>16</sup>

## Symptoms

The major symptom of spondylolisthesis is low back pain. It often radiates into the sacroiliac region but also into the region of the thighs and distally. It usually does not have a specific dermatomal pattern and often is a paresthesia rather than pain.

Examination reveals organic signs of limited flexibility and a palpable "ledge" may be felt at the upper aspect of the listhesis. There may be a segmental lordosis, which when aggravated by the examiner causes or increases the pain. On rectal examination, the prominent sacrum may be palpated as a "mass."

Limited hamstring extensibility is often associated with spondylolisthesis. A muscular positive straight leg raising test is elicited. The cause of limited hamstring elongation remains unconfirmed, as electromyographic studies have failed to discern a neurological basis.<sup>17-19</sup> A

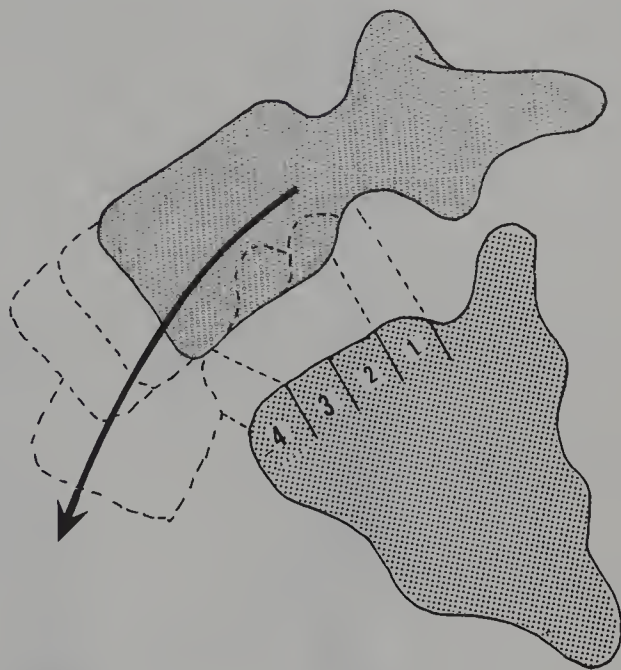


Figure 11-5. Grading of spondylolisthesis. There are four grades of spondylolisthesis of lumbar five on the sacrum. Grade four reveals forward slipping off the sacrum.

waddling gait has been described but is not a characteristic positive sign.

Radiological signs are well documented. The degree of listhesis has been documented (Fig. 11-5).

## Treatment

In a long (20-year) study<sup>20</sup> adolescents progressed 2.5 mm and adults 5.0 mm. Spondylolisthesis at L4-5 progressed farther than listhesis at L5-S1. Disk degeneration was noted at the initial evaluation in 40% of patients, with increase to 50% at L5-S1 and 70% at L4-5. Ninety-one percent of patients had low back pain, usually considered as chronic. Sciatica was reported in 25% of patients.

Treatment depends on the severity of symptoms and their resultant impairment and disability and confirmation that the listhesis is the major cause of the symptoms. The lordosis must be decreased to reduce the gravitational forces on the superior vertebra sliding on the inferior vertebra.

A brace (Fig. 11-6), corset, or cast that decreases the lordosis (Fig. 11-7) and maintains the gained position is both diagnostic and therapeutic. Exercises to decrease the lordosis and strengthen the muscles involved must be instituted and supervised whether a support is pre-



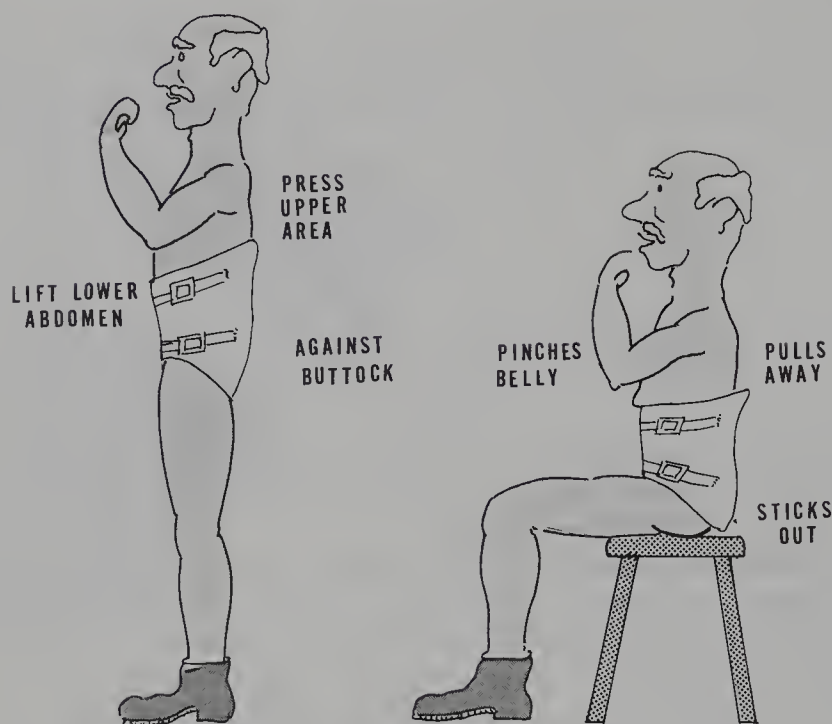


Figure 11-6. A corset in the standing and sitting position. The upright bracing presses against the buttocks and the lower thorax, uplifting the abdomen. This ensures a decrease of the lordosis. The brace in the seated position often fails as the contact points are lost and the anterior portion wrinkles and pinches the abdomen.

scribed or not. In patients considered not surgical candidates and treated with exercise,<sup>21</sup> flexion type exercises were less likely to require use of back supports, require job modification, or limit their activities because of pain.

The isthmic type of listhesis is a fatigue fracture that often heals by fibrous tissue. When noted as an acute fracture, as indicated by x-ray and a bone scan, the fracture usually heals with rest and time. A corset gives immobilization during the time.

When a pars interarticularis defect is seen in an adolescent, athletic activities should be curtailed. Abdominal muscle strengthening exercises should be initiated in all patients with a lysis or a listhesis.

Surgical intervention is indicated usually by presence and progression of neurological symptoms and, conceivably, when a cast benefits the patient subjectively to relieve the pain. Predictors of surgical intervention are often based on the sagittal rotation of the infection vertebra (Fig. 11-8) as well as the degree of listhesis. Predictors are a greater than 50% slip, with increasing sagittal posterior rotation of the sacrum and anterior rounding of the superior surface of the caudal vertebral body.<sup>22</sup>

Figure 11-7. Principles of bracing. The lumbosacral corset has the purpose of decreasing the lordosis, reinforcing weak abdominal muscles, and restricting active and passive motion. (1) Uplifts the abdomen and replaces abdominal muscle strength. (2) The upper aspect of the brace in the corset reaches above the lumbar spine (T12). (3) The lower portion of the corset stays pressed against the sacrum to decrease angulation and cause the sacrum to be more vertical. A corset should always be accompanied by appropriate flexion exercises, training in posture, and proper use of the low back in daily activities.

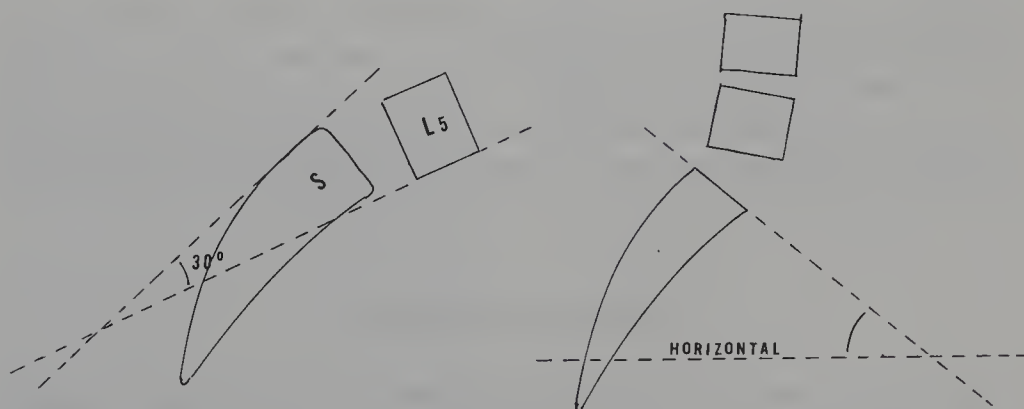
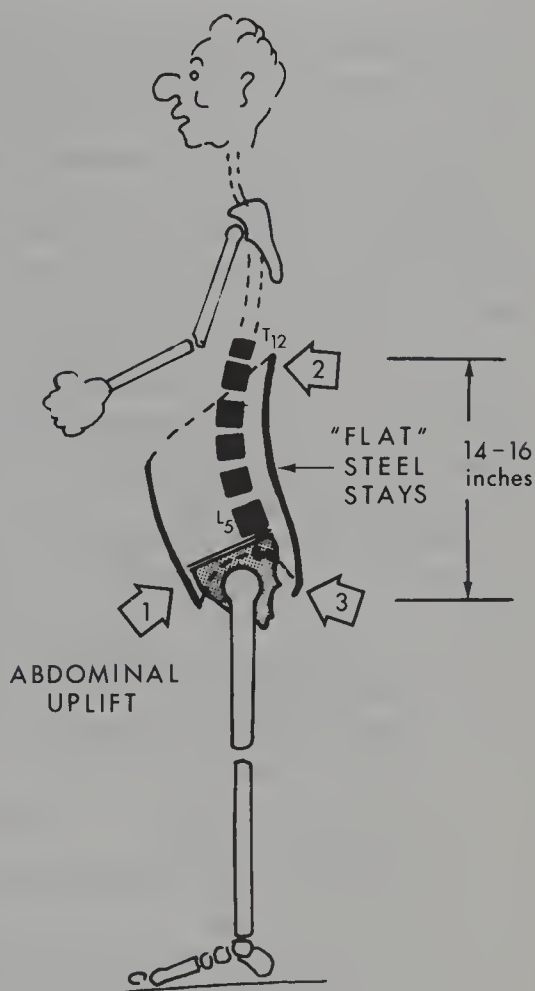


Figure 11-8. Radiologic predictors of surgical needs. The left figure depicts sagittal rotation and the right illustration depicts the sacroiliac angle in the upright position.

Patients treated surgically as compared with those treated nonsurgically<sup>23</sup> seemed to benefit equally. In situ fusion for persistent pain or neurologic dysfunction also had good long-term results so the answer as to surgery or no surgery remains unconfirmed.

The specific techniques have had voluminous literature and will not be discussed here, as this text relates to conservative care, and surgery is not the expertise of the author. The standard technique of spinal fusion<sup>24</sup> is predicated on the diagnosis of spinal instability.<sup>25</sup> Posterior decompression of the neural arch or the isthmic defect is directed to remedy spinal stenosis secondary to the listhesis. Reduction of the listhesis from surgery is unpredictable, and fusion in situ usually has limited success. Neurological decompression appears justified, but even that remains unconfirmed.

The following trends appear in the literature as predictors of postoperative instability as stated by Garfin and Herkowitz.<sup>25</sup>

1. The larger the preoperative slip, the greater the risk of postoperative slippage.
2. Postoperative slip does not necessarily correlate with development of symptoms.
3. Decompression across disk space of normal height may lead to postoperative slip.
4. Excision of over 50% of both facets at the same level increases the risk of slip.
5. Increasing the levels of decompression increases the risk of slip.
6. Penetration of the disk space at a decompressed level increases the risk of slip.
7. Women are at a higher risk than men for development of postoperative slip.

With predictor 2 stating that postoperative slip does not necessarily correlate with development of symptoms, one can wonder why the other predictors occur. The value of surgery is also questionable and currently not a totally acceptable procedure, so the proper conservative treatment of flexion exercises, corsetting, and back school training with careful monitoring appears to be the ideal approach.

## SPINAL STENOSIS

Lumbar spinal stenosis has been defined as a condition involving any type of narrowing of the spinal canal, nerve root canals, or tunnels of intervertebral foramina.<sup>26</sup> The narrowing not only reduces the anteroposterior and lateral diameters but also alters the cross-sectional

configuration of the spinal canal. The precise determination of what constitutes stenosis relies exclusively on radiological measurement, cadaver dissections, or operative observations.

The condition termed *spinal stenosis* was initially described by Verbiest<sup>27</sup> and was divided into absolute stenosis of the lumbar spine (ASLC) or relative stenosis of the lumbar spine (RSLC). The definition of narrow canals varied with the examiner, with the predominant rating being canal to body: A-P diameter.<sup>28</sup> An A-P diameter less than 10 mm was considered pathological,<sup>29</sup> although numerous authors have given varying numbers, with 12 mm being the most consistent.

The normal lumbosacral canal is narrowest in its anteroposterior diameter at the third and fourth lumbar vertebra, and its size increases caudally. At L5-S1 the shape of the canal is trefoil. The measurement of the canal is purely an indication of potential for the development of symptoms and does not reveal the site of the stenotic symptoms.<sup>30</sup>

The dorsolateral wall of the spinal canal is formed by the L5 lamina and superior facet of the sacrum, while the ventrolateral wall is the superior facet of the sacrum and the inferior facet of the fifth lumbar vertebra that forms the entrance of the root canal (foramen) (Fig. 11-9).

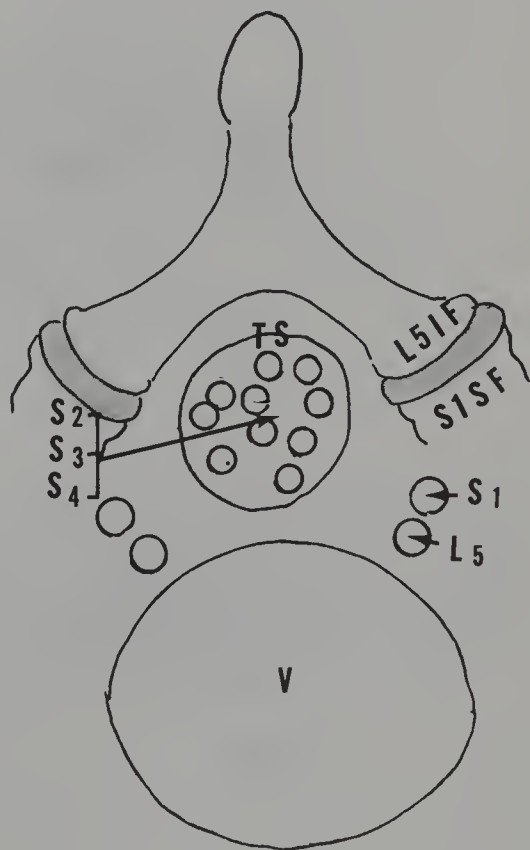


Figure 11-9. Spinal canal contents. The posterior wall of the canal comprises the lamina, which form the facets. At the L5 to S1 joints the L5 inferior facet (L5IF) articulates with the superior facets of the sacrum (S1SF). The thecal septum (TS) encloses the S2, S3, and S4 nerve roots and the L5 and S1 roots pass within the foramen between the facets and the vertebral body (V).

Spinal stenosis can be considered as congenital or developmental, with the latter being subdivided into idiopathic or achondroplastic. Acquired stenosis is further subdivided into degenerative (central, peripheral, or spondylolisthesis), iatrogenic (postlaminectomy, postfusion, postchemonucleolysis), post-traumatic or secondary to systemic disease such as Paget's.<sup>31</sup> Narrowing is also considered as secondary to disk herniation.

Narrowing can occur in one or several locations of the same vertebral segment or can affect several segments with similar changes. The size of the canal can be decreased by soft tissue changes or bony changes such as hypertrophy of the facets (Fig. 11-10), by posterior bulging of the disk, or by hypertrophic degenerative spurs of the vertebrae (Fig. 11-11). Spinal stenosis also occurs secondary to structural scoliosis (Fig. 11-12).

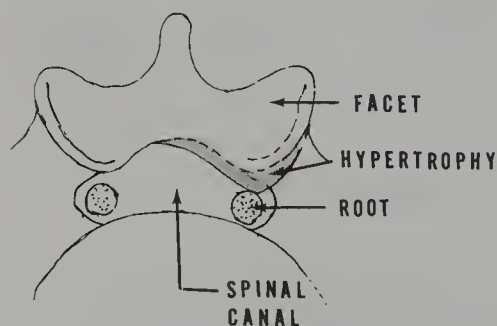
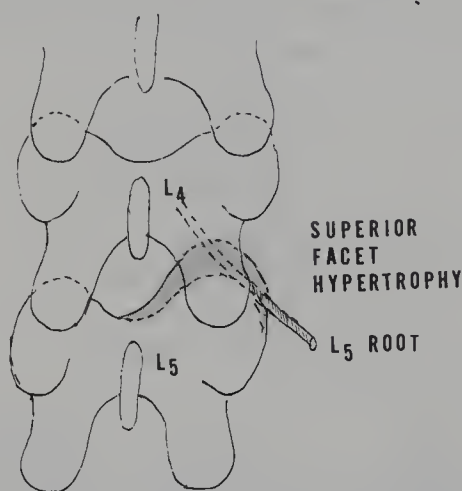


Figure 11-10. Spinal stenosis from superior facet hypertrophy. The *upper figure* (posterior view) shows hypertrophy of the superior facet of L5 (fifth lumbar vertebra) encroaching on the foramen, entrapping the L5 nerve root (foramenal stenosis). The *bottom figure* (superior view) shows how the hypertrophied facet compresses the nerve root.



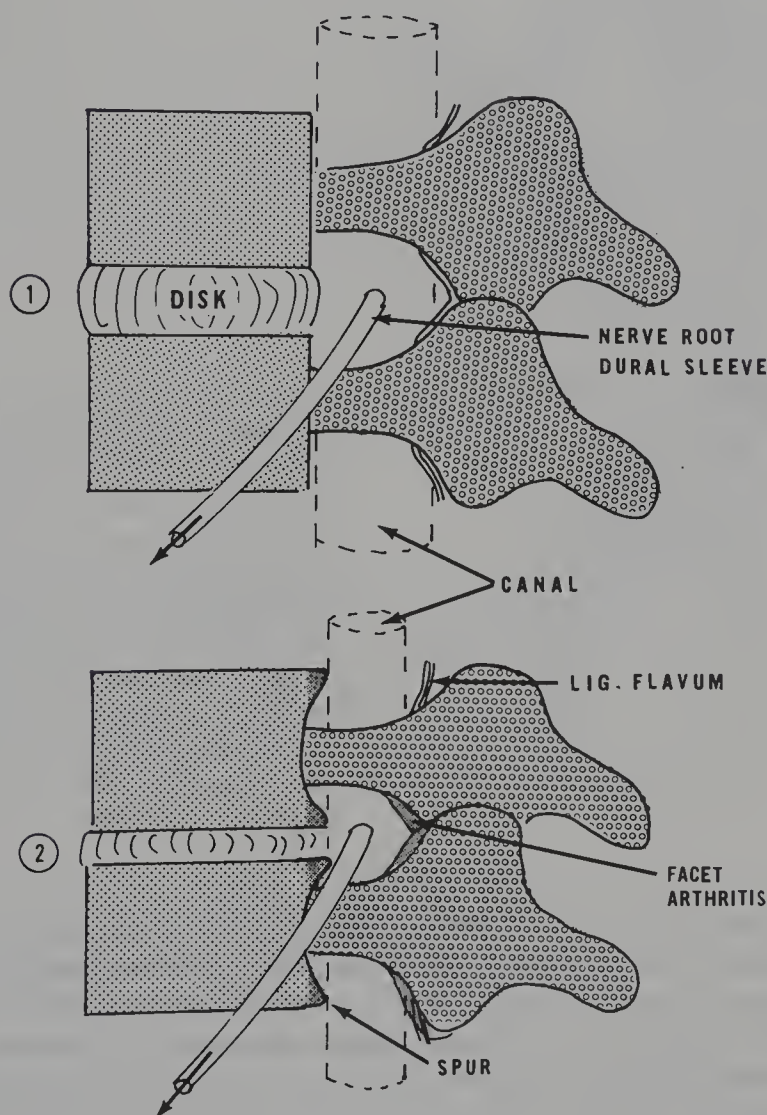


Figure 11-11. Spondylosis and spinal stenosis. (1) A normal functional unit with a normal spinal canal (*dotted lines*) and normal foramen through which emerge the nerve root in its dural sleeve is depicted. (2) Degenerative disk disease with osteophytes encroaching on the foramen and the canal is depicted. The ligamentum flavum hypertrophies, causing further spinal stenosis, and facet arthritis encroaches both on the canal and the foramen.

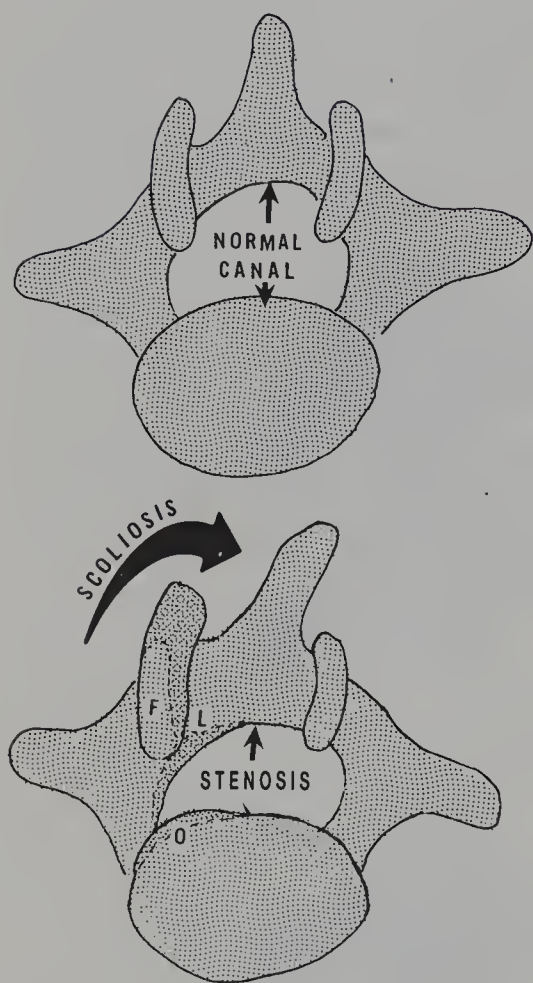


Figure 11–12. Stenosis secondary to structural scoliosis. *Upper view* shows a normal functional unit with a normal canal. Structural scoliosis deforms the vertebra with hypertrophy of the lamina (L) and hypertrophy of the facet (F) on the convex side. As there is also variance of the disk space osteophytes (O) also occur with scoliosis. These changes encroach on the canal and the foramen. The *curved arrow* shows the direction of the scoliosis.

Reduced space of the lumbosacral canal or dural space produces neurogenic syndrome only if the nerve roots are excessively stretched by the stenosing material. The vascular arrangements of the nerve roots are highly vulnerable to compression or traction. The veins consist of complicated sinusoidal channels.<sup>32</sup> Exercise of a limb has been shown to dilate the blood vessels of the emergent nerve roots within the foramina canals.<sup>33</sup>

When we walk, the roots become more taut on flexion and relaxed on spinal extension (Fig. 11–13), but on extension they increase their cross section and the foramina narrow. These changes in the spinal canal (Fig. 11–14) and the dura (Fig. 11–15) are similar to those that occur in the cervical spine.<sup>34</sup>

It is apparent, therefore, that it is not only the mechanical stenosis that produces symptoms but also the spinal movement. Gross lumbar spondylosis with associated rigidity is usually not associated with claudication symptoms. Exertional pain in the leg or thigh has been asso-

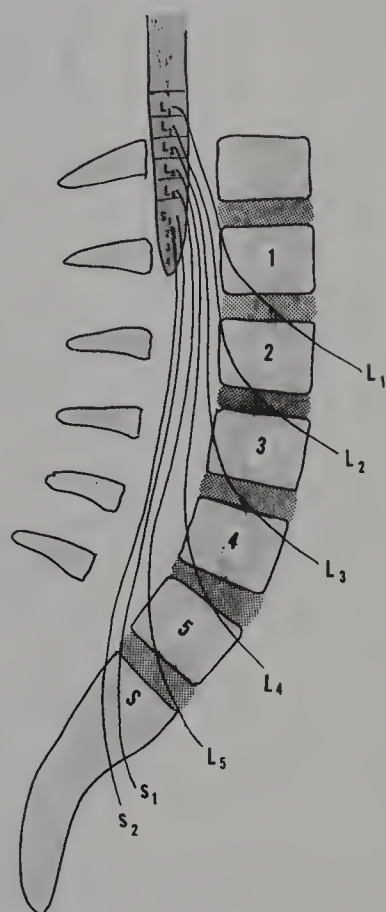


Figure 11-13. Nerve roots in neutral lordosis. The normal lordosis shows the nerve roots (L1, L2, L3, L4, L5, S1, and S2) emerging from the caudal equina as being relaxed as they descend to their foramina.

ciated with occlusive arterial disease but also has been shown to occur from compression of the cauda equina.

The clinical condition considered as a sequela of spinal stenosis (ASLC or RSLC) was termed *neurogenic claudication* due to compression of the nerves of the cauda equina.<sup>27,28</sup> The symptoms of neurogenic claudication were depicted as pain, paresthesias, or impairment of sensory or motor power noted in the leg(s) on walking or standing. Major symptoms are usually subjective and vague and difficult to objectively substantiate. Back pain often exists, with leg pains occurring later. Back symptoms are stiffness on awakening, lessening with activity but aggravated by prolonged standing and walking. Sleeping postures are impaired with prone laying discomfort and relief from assuming the fetal position.

Leg symptoms often are stated as a sensation of coldness, tingling, "falling asleep," or vague weakness. Symptoms usually occur in both legs but can occur in one leg. Ataxia develops after walking in some cases but only in cases involving both legs.<sup>35-39</sup>

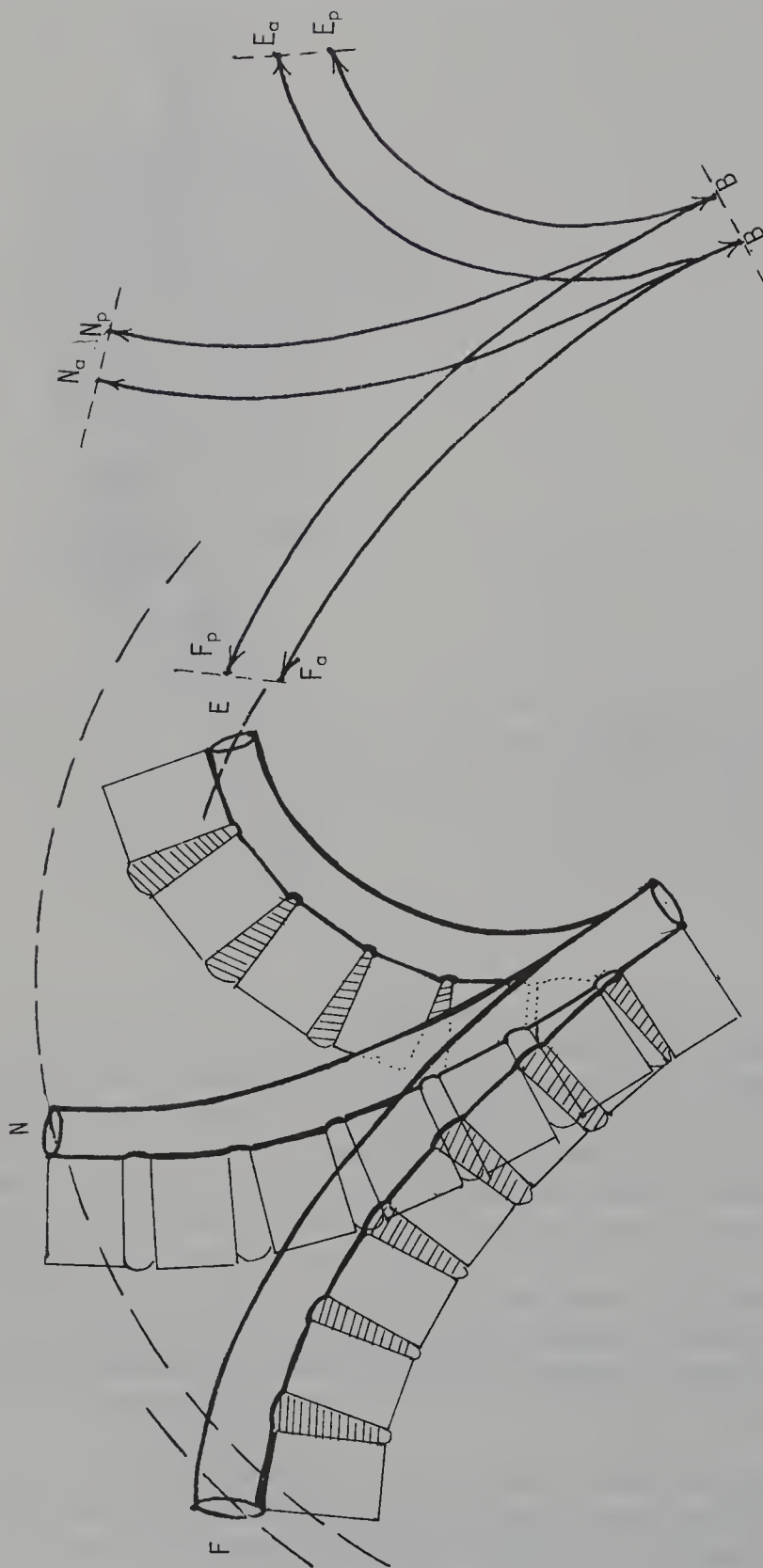


Figure 11 - 14. Alterations in spinal canal length. The *left figure* shows the canal in flexion (F), neutral (N), and extension (E). The length of the canal (*right figure*) shows a lengthening of the canal on flexion posterior wall length (Fp) and anterior wall length (Fa). In the neutral position the wall lengths are depicted: anterior wall Na and posterior wall Np. On extension the canal shortens. The anterior wall (Ea) and posterior wall (Ep) are shorter.

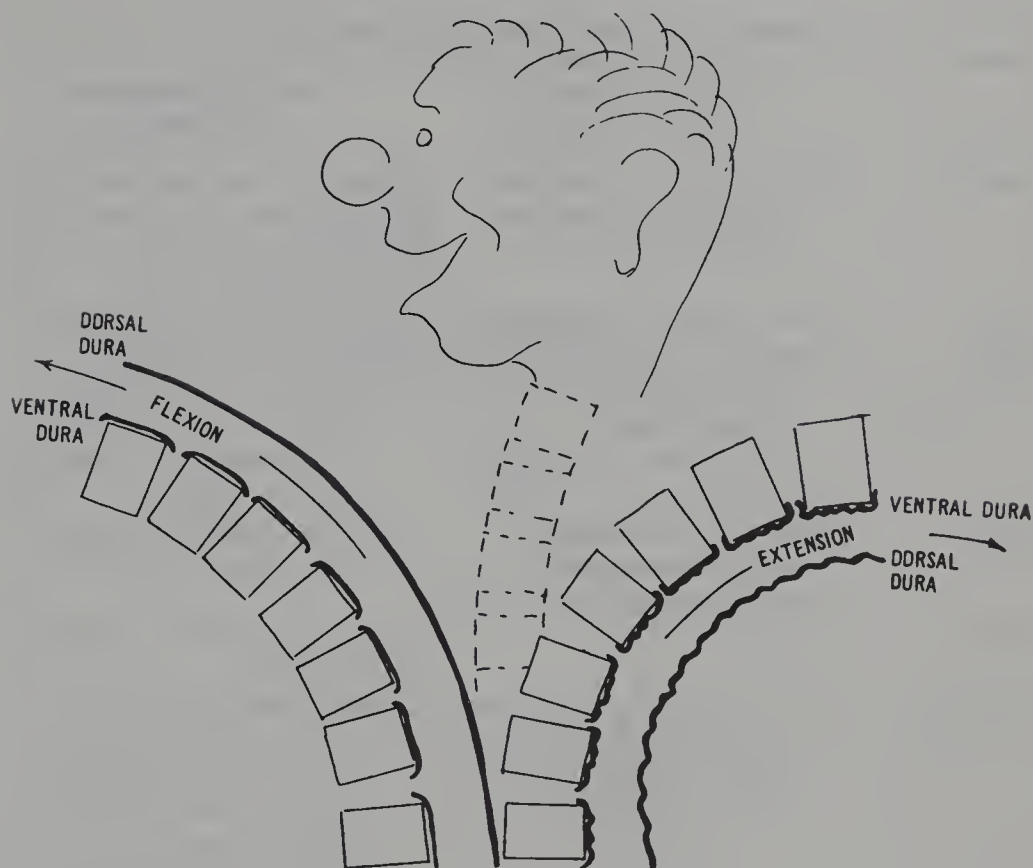


Figure 11-15. Reaction of the dura in flexion and extension. On spinal flexion the dura is elongated whereas on extension it plicates.

What classically designates the condition is the syndrome of pseudoclaudication. Walking or leg exercises give symptoms of vascular claudication in that the symptoms occur and increase with exercise and intensify with prolonged exercise. Unlike vascular claudication (hence the prefix *pseudo*), the pain does not cease from merely stopping exercise but ceases or diminishes in stopping *and* assuming a flexed posture of the lumbosacral spine. Lower extremity pulses are also not diminished as in vascular claudication (Table 11-1).

Neurological findings are sparse, so pseudoclaudication remains a subjective diagnosis alluded to as being spinal stenosis *if* radiological findings demonstrate measurable stenosis.

Treatment should be conservative, with surgery considered only in the advent of intolerable and disabling pain denying functional activities of daily living or progressive neurological deficit in spite of adequate nonoperative treatment.

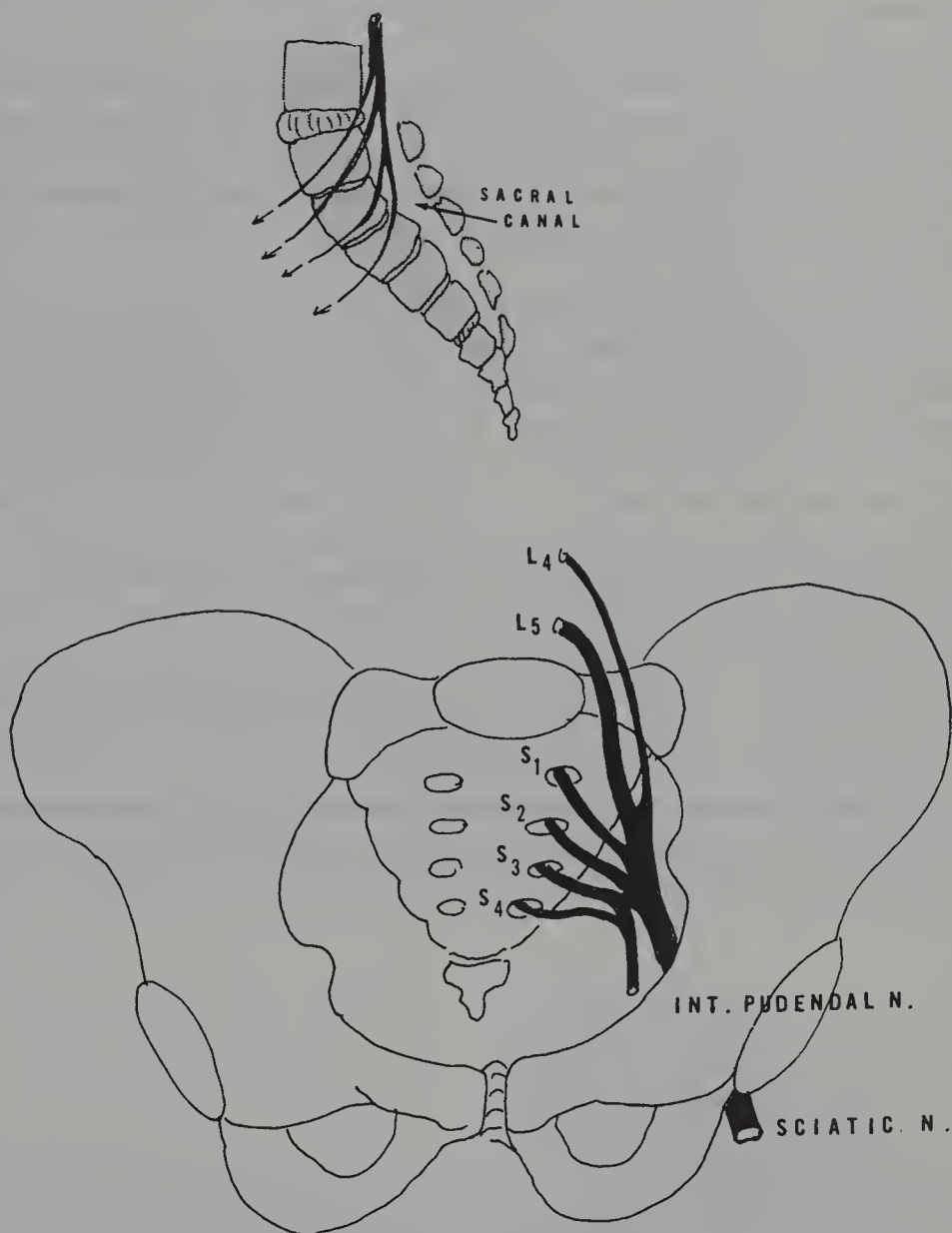


Table 11-1. DIFFERENTIAL DIAGNOSIS

	<i>Aortoiliac Occlusive Vascular Disease</i>	<i>Pseudoclaudication/ Spinal Stenosis</i>
Pain site	Hips, thighs, and buttocks, progressing to calf (or in reverse direction).	Lumbar spine and buttocks. Pain may occur from prolonged standing or lying with back arched.
Onset	After walking a long distance.	After walking up inclines.
Pain description	Aching, squeezing, cramping quality increases with walking. Rarely paresthesias or weakness.	Onset may be tingling, weakness, or clumsiness. Pain may be minimal, often numbness or burning, not cramping.
Relief	Fast relief by ceasing to walk. Slowing walk decreases onset or severity.	Must sit down and assume flexed trunk posture.
Impotence	Frequent.	May take 20 to 30 minutes for relief.
Peripheral pulses	May disappear after walking. Loud bruits, frequently pale legs after walking, no neurologic findings.	Saddle distribution of numbness frequent. SLR may not be present, and cough may not increase radicular pain. After prolonged walking, pain may occur from SLR. Ankle jerk may be absent after exercise.

Conservative treatment implies achieving a flexed lumbosacral spine, that is, decreasing the lumbar lordosis. Trunk flexion exercises already stated in this text must be instituted and continued. Extension exercises advocated in herniated disk disease must be avoided. Strong abdominal muscles must be gained and everyday postures corrected. Pelvis traction may afford brief transient relief and is worthy of trial. Epidural steroids are also transient in their relief.

The various techniques of surgical intervention are beyond the scope of this text, but they vary with the pathology causing the stenosis. Surgery alleviates the compression on the neural canal contents (Fig. 11-16) and does not basically alter the original pathology. The axiom of spinal surgery must be observed: the more precise the location and origin of the site of pain, the better the predictable end result.



**Figure 11-16.** Nerve roots emerging from the lumbosacral spine forming the sciatic nerve. The sacral plexus formed from L4, L5, S1, S2, S3, and S4 nerve roots divides to form the sciatic nerve, the pudendal, and the posterior cutaneous nerve of the thigh (not shown).

## PIRIFORMIS SYNDROME

This is a controversial syndrome that implies compression of the extraspinal nerves (Fig. 11-16), forming the sciatic nerve by the piriformis muscle.

The sciatic nerve passes through the greater sciatic foramen in close proximity to the piriformis muscle (Fig. 11-17). The fibro-osseous tunnel through which the nerve passes is the site of compression.<sup>40</sup> The sacrospinal and sacrotuberous ligaments connect the ischium to the sacrum and delineate two foramina: the greater and the lesser (Fig. 11-18).

The piriformis muscle splits the foramen into the suprapiriformis and the infrapiriformis regions. The infrapiriformis foramen is triangular in shape, with the superior margin being the inferior margin of the piriformis muscle and the inferior margin being the sacrospinalis ligament. Laterally, the margin is the bony margin of the sciatic notch. Two neurovascular bundles leave via this notch: the pudendal neurovascular bundle medially and the sciatic nerve laterally. Occasionally (21% of patients) the nerves emerge through a division in the piriformis.<sup>41</sup>

The ligaments of the pelvis are involved in the angulation of the sacrum (Fig. 11-19).

The size of the belly of the piriformis varies greatly, thus varying the opening of the foramen. In 50% of the population, a synovial bursa exists between the muscle and the bone.

The piriformis muscle is an external rotator as well as an extensor of the hip. In the flexed hip position, the muscle abducts the hip.

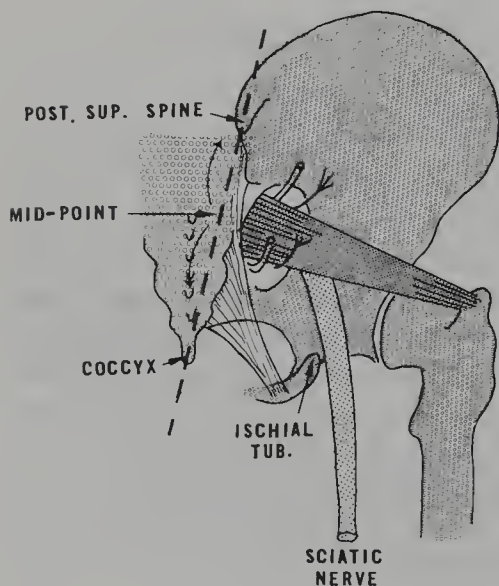
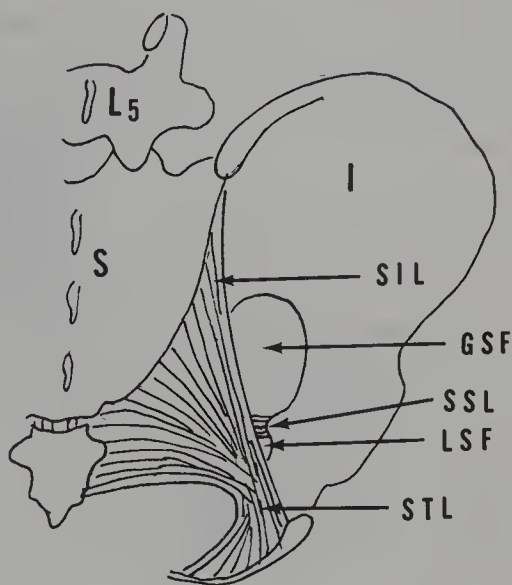


Figure 11-17. Piriformis muscle. The piriformis muscle and its anatomic relationship to the sciatic nerve is shown.

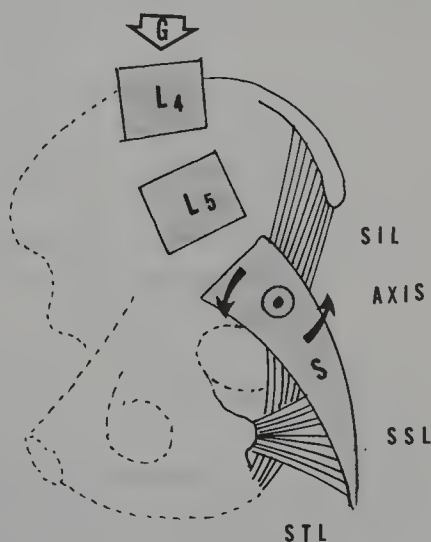
**Figure 11-18.** Ligaments of the pelvis: posterior view. The ligaments of the pelvis are shown from a posterior view. L5 is the fifth lumbar vertebra, I is the ilium, S is the sacrum, SIL is the sacroiliac ligament, SSL is the sacrospinous ligament, and STL is the sacrotuberous ligament. GSF is the greater sciatic foramen and LST the lesser sciatic foramen.

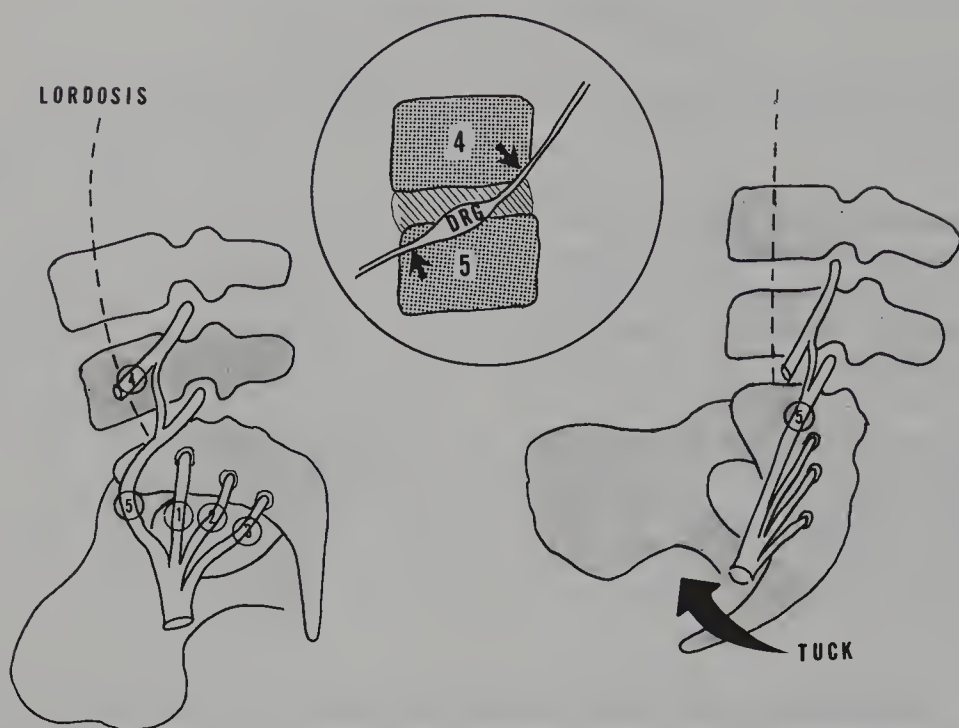


When a straight leg raising test is performed at  $20^\circ$ , the piriformis is stretched.<sup>42</sup> The nerve roots forming the sciatic and sacral nerves vary in their elongation as regards to the angulation of the sacrum. In the lordotic position (Fig. 11-20), the nerves are slack, only to be under tension in a decreased lordosis.

Increased tension of the pelvis-femoral muscles develops as they attempt to stabilize the pelvis. These muscles, of which the piriformis is one, hypertrophy. The foramina do not significantly vary in their opening, so nerve compression or traction must occur distal to the foramina.

**Figure 11-19.** Ligaments that resist rotation of the sacrum. G is the force of gravity. L4 and L5 are the two lower lumbar vertebrae and S is the sacrum. The *small round circle* is the axis of rotation of the sacrum as depicted by the two *curved arrows*. SIL = sacroiliac ligament; SSL = sacrospinous ligament; and STL = sacrotuberous ligament. By their lines of attachment the resistance to rotation of the sacrum about the axis is apparent.





**Figure 11-20.** Nerve root direction modification with angle of the sacrum. The *circled drawing* depicts the relationship of the dorsal root ganglion (DRG) to the disk between L4 and L5. The *left drawing* shows lumbar lordosis with the nerve roots relaxed. The *right drawing* depicts that with the pelvis tucked the lumbar spine becomes kyphotic with nerve roots taut.

The mechanism by which the sciatic nerve is entrapped by the piriformis within its osseo-fibrous-muscular compartment remains obscure. Flexion contracture of the hip tends to chronically increase and maintain a lordosis that will increase compression of the neurovascular bundle. The postulated etiologies of the piriformis syndrome are:<sup>42</sup>

1. Sacroiliac disease that causes muscle contraction of the piriformis muscle
2. Inflammatory disease of the muscle, tendon, or fascia of the piriformis
3. Degenerative deformities of the bony component of the notch
4. Abnormalities of the neurovascular bundle as they course through the tunnel
5. Direct trauma to the gluteal region (gluteus maximus) or sacroiliac joint<sup>43-47</sup>

## Clinical Symptoms and Signs

Differentiation of sciatica from lumbar discogenic disease, foraminal stenosis, or even vascular etiology must be ascertained. Pain or



paresthesias may be present along the entire distribution or a segment of the sciatic nerve. Paresthesia may be a burning sensation, hyperesthesia, or even anesthesia. Motor deficit may co-exist with subtle atrophy. From sympathetic nerve involvement, trophic changes may occur in the affected dermatomal skin area.

Pain in the sacral and gluteal areas<sup>48</sup> is a predominant symptom, which increases with sitting and walking and decreases from assuming the supine position.

Sciatica from the piriformis, differing from discogenic neuritis, is indicated by the performance of straight leg raising with the foot in a straight coronal position and then the performance of straight leg raising with leg (foot) actively, internally rotated. The rotation demands contraction of the piriformis. In the seated position, this test of actively, internally rotating the leg also requires active abduction of the upper leg done by contraction of the gluteal muscles as well as the piriformis. This is termed the *Gower-Bonnet test*.<sup>42</sup>

Rectal examination reveals palpable tenderness and swelling of the piriformis muscles. This rectal examination also eliminates pelvic pathology that may mimic the syndrome.

## Treatment

Conservative treatment consists of pelvic tilting exercise (Fig. 11-21), stretching the contracted hip flexor(s), and oral anti-inflammatory

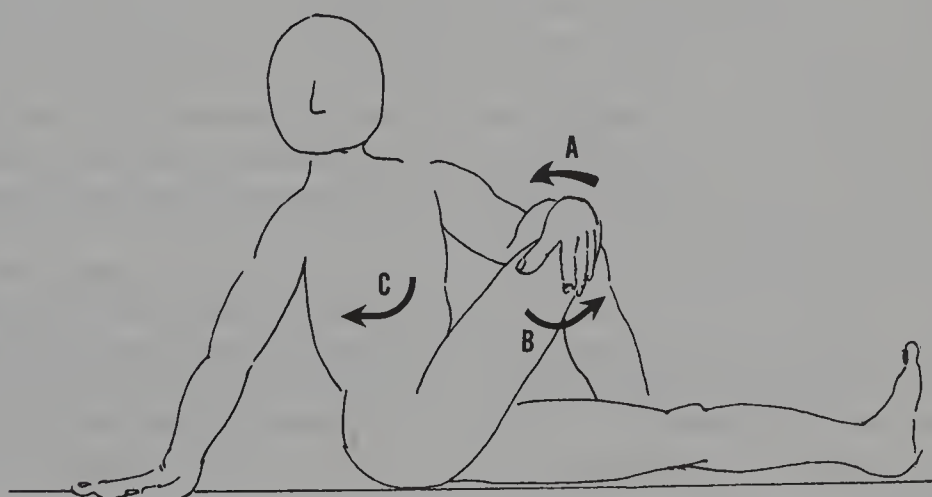


Figure 11-21. Piriformis test and treatment. When the piriformis is inflamed or in spasm it will hurt when stretched as in the illustration. To reduce spasm gentle stretching in the same manner gradually decreases the spasm. The foot on the side of inflammation is placed at the knee level on the other side of the extended leg and the leg is pushed towards the floor and up (A and B) and the trunk turned in the opposite direction (C).

medication. Injection of an anesthetic and steroid into the piriformis bursa or even into the piriformis muscle has its advocates. This injection can be administered in one of two ways: vaginally by direct insertion into the tender muscle or via the gluteal muscle aiming at the sacral notch toward the palpably located tender spot located by the digital rectal examining finger.

If the syndrome persists over 6 months without abating, surgical sectioning of the piriformis tendon is advisable. The section does not cause any noticeable functional loss of the hip.<sup>49</sup>

## SACROILIAC PATHOLOGY CAUSING LOW BACK PAIN

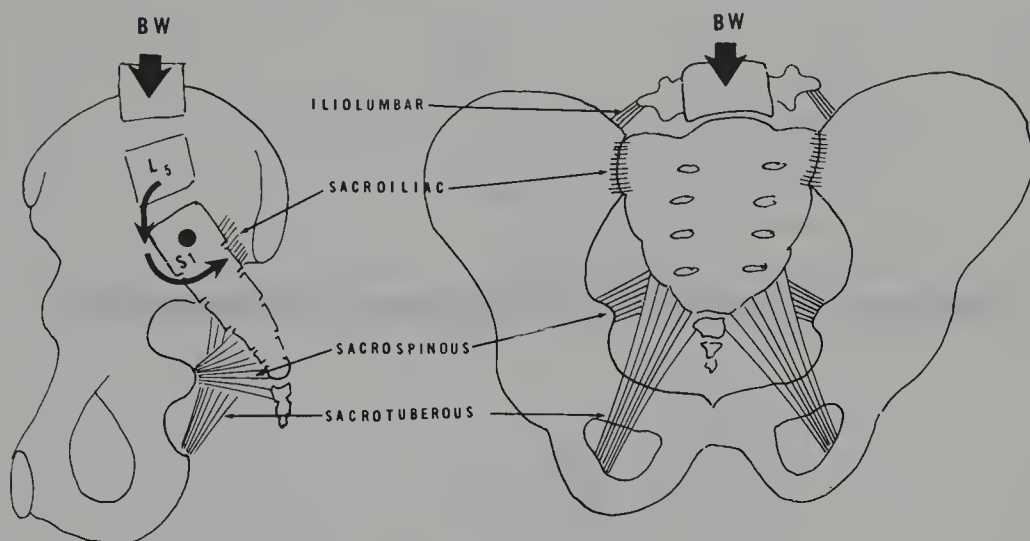
The sacroiliac joint is the *bête noire* of low back pain, with enthusiastic proponents and equally vociferous opponents. Goldthwaite and Osgood<sup>50</sup> introduced the condition of "sacroiliac strain," which led to numerous diagnostic procedures and therapeutic approaches in this etiology of low back pain.

In 1970 Bordillon,<sup>51</sup> a proponent of sacroiliac pathology and also an expert in the modality of manipulation, stated, "The range of motion of the sacroiliac joint is small and demonstration by direct palpation is both difficult and unconvincing. . . . Indeed the existence and importance of sacroiliac mobility is still a subject of argument." Movement clinically determined, however, has been claimed.<sup>52</sup>

Unless there is demonstrable pathology or subluxation noted on radiological studies, pathology existing there is questioned. The sacroiliac joint is an extremely stable joint by virtue of its incongruous articular surfaces and the presence of powerful ligaments located both anteriorly and posteriorly (Fig. 11-22).

As Mooney<sup>53</sup> says, "It is difficult to separate pain emerging from the sacroiliac joint from pain emerging from the facet joints," because there is overlapping innervation. The test for sacroiliac pathology termed the *Gaenslen sign* implied reproduction of low back pain from unilateral hyperextension of the hip, but this has been refuted as being specific as it also hyperextends the low back, impinging the facets.

Degeneration occurs in the sacroiliac joints, with some estimates<sup>54</sup> stating evidence in 67% of patients older than 55 years of age, implying some movement must exist in spite of no radiologic evidence of movement between symptomatic and asymptomatic patients.<sup>55</sup>



**Figure 11-22.** Ligamentous support of the pelvis. *Left*, A side view of the pelvis indicates the body weight (BW) on L5 vertebra causes rotation of the sacrum (*curved arrows*). This rotation is resisted by the sacrospinous and sacrotuberous ligaments. *Right*, these two ligaments and ligaments that connect the sacroiliac joint and connect the transverse processes of the fifth lumbar vertebra are depicted. This last ligament has been considered a site of pain in low back pain syndromes.

## Diagnosis of Sacroiliac Pathology

Movement of the sacroiliac joint is attempted to ascertain that *that* movement causes low back pain in the area of the suspected joint. With the patient in the supine position on a firm surface, the examiner pushes down firmly on both anterior superior spines. Pressure is then exerted on one anterior superior spine to rotate the pelvis. In the same supine position, the examiner places both hands on the outer rims of the pelvic rims and presses them together. In the same position, the pelvis is firmly held by the examiner's hands and is rocked in a rotary manner. Resisting abduction of the extended leg also is considered to initiate movement of the sacroiliac joint (Fig. 11-23).

Diagnosis of pain emanating from the sacroiliac joint has been postulated from benefit by injecting an anesthetic agent into the joint under fluoroscopic control.<sup>56</sup>

## Treatment of Sacroiliac Pathology

Oral anti-inflammatory agents combined with local applications of ice and then heat are the initial efforts. A compressive pelvic belt is also

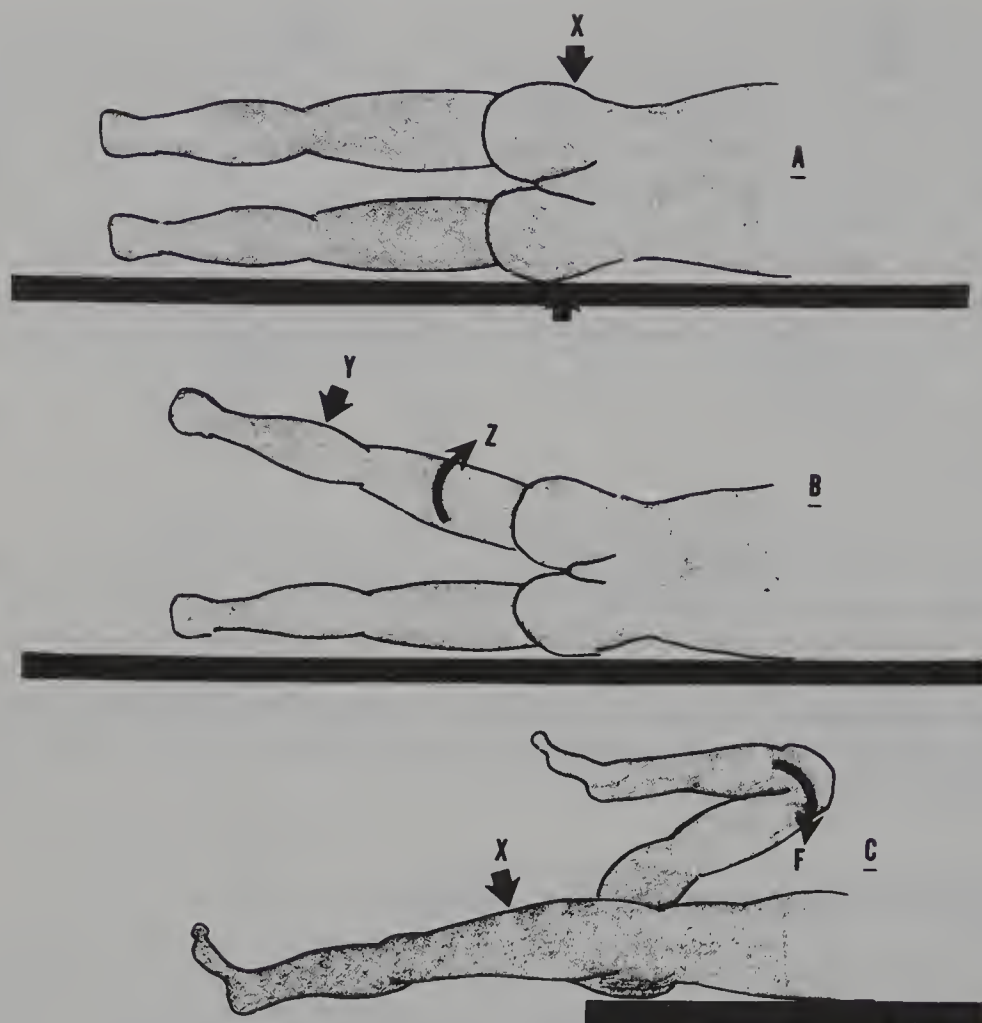
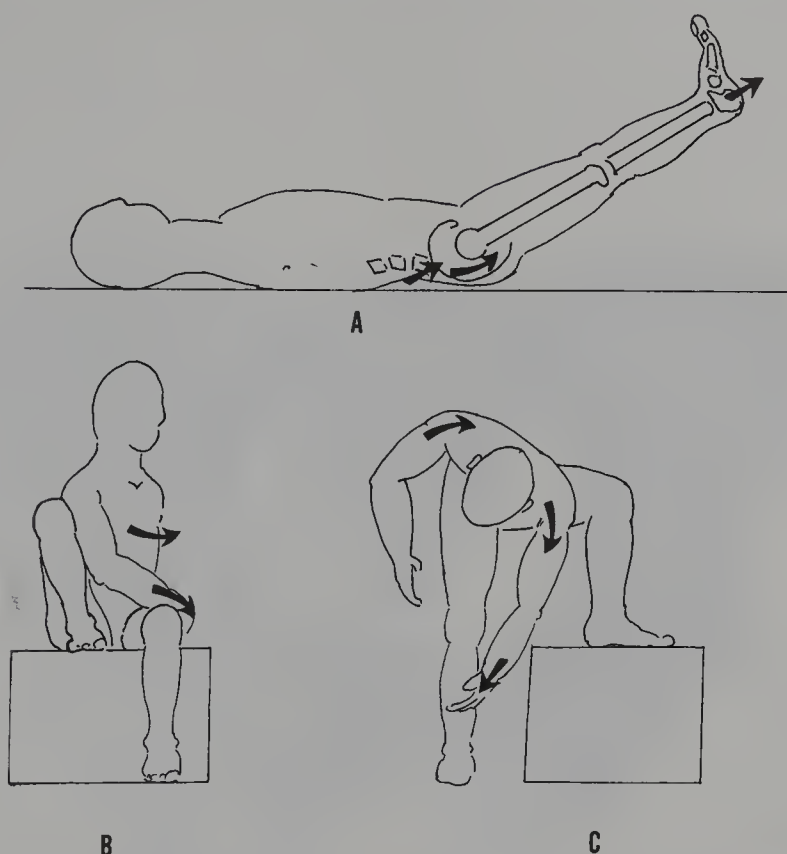


Figure 11-23. Sacroiliac tests. (A) Upper figure shows that direct lateral pressure on the iliac crest with patient supine may cause S1 pain. (B) Middle illustration shows test by having supine patient abduct and extend the leg (Z) against resistance (Y). (C) With patient supine and one leg fully flexed against the chest (F) the other dependent extended leg is hyperextended (X). This allegedly initiates sacroiliac pain and by flexion one leg eliminates lumbosacral facet pain.

advocated. Local injection of analgesia and steroids into the affected joint has benefit. Self-mobilization exercises attempt to correct the dysfunction. Pelvic mobilization exercises are indicated, as is correction of faulty posture.

Passive elongation of the affected leg reportedly is beneficial as are the rotational exercises demonstrated in Figure 11-24.



**Figure 11-24.** Sacroiliac exercises. (A) Traction of one leg with patient supine rotates the pelvis (*curved arrow*). (B) Seated patient uses arm to rotate trunk (*lower curved arrow*) and also actively rotates the trunk (*upper curved arrow*). (C) Standing with one leg on bench patient actively rotates pelvis downward.

As stated, injection of analgesic and steroids into the joint is therapeutic as well as diagnostic and is preferably performed under fluoroscopic viewing. Surgical fusion is indicated if severe, significant, and symptomatic subluxation of the sacroiliac joint is radiologically demonstrable.

### Sacroiliac Pathology Secondary to Pelvic Fracture

A pelvic fracture frequently results in sacroiliac dysfunction, subluxation, and even luxation. If a pelvic fracture causes any change in the pelvic ring (Figs. 11-25 and 11-26), radiologic studies should be carefully evaluated for sacroiliac pathology. Routine x-rays may fail to



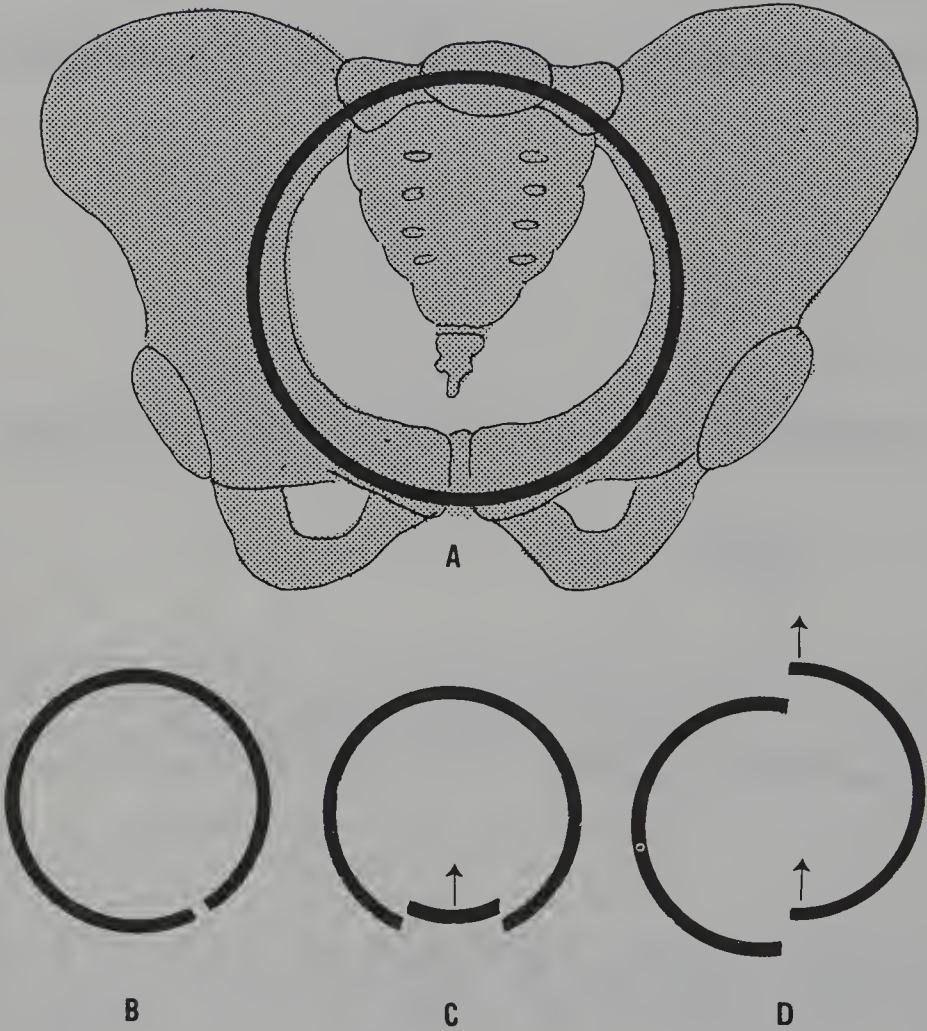


Figure 11-25. Pelvic ring. The pelvic ring is bounded laterally by the ilia, anteriorly by the pubic rami, and posteriorly by the sacrum and the sacroiliac joints. The *upper figure* (A) shows an intact ring. (B) A pelvic fracture **without** disruption of the ring. (C) A subluxation of a segment of the ring **without** disruption of the ring. (D) A total disruption of the ring, both the symphysis pubis and the sacroiliac joint (*small arrows*).

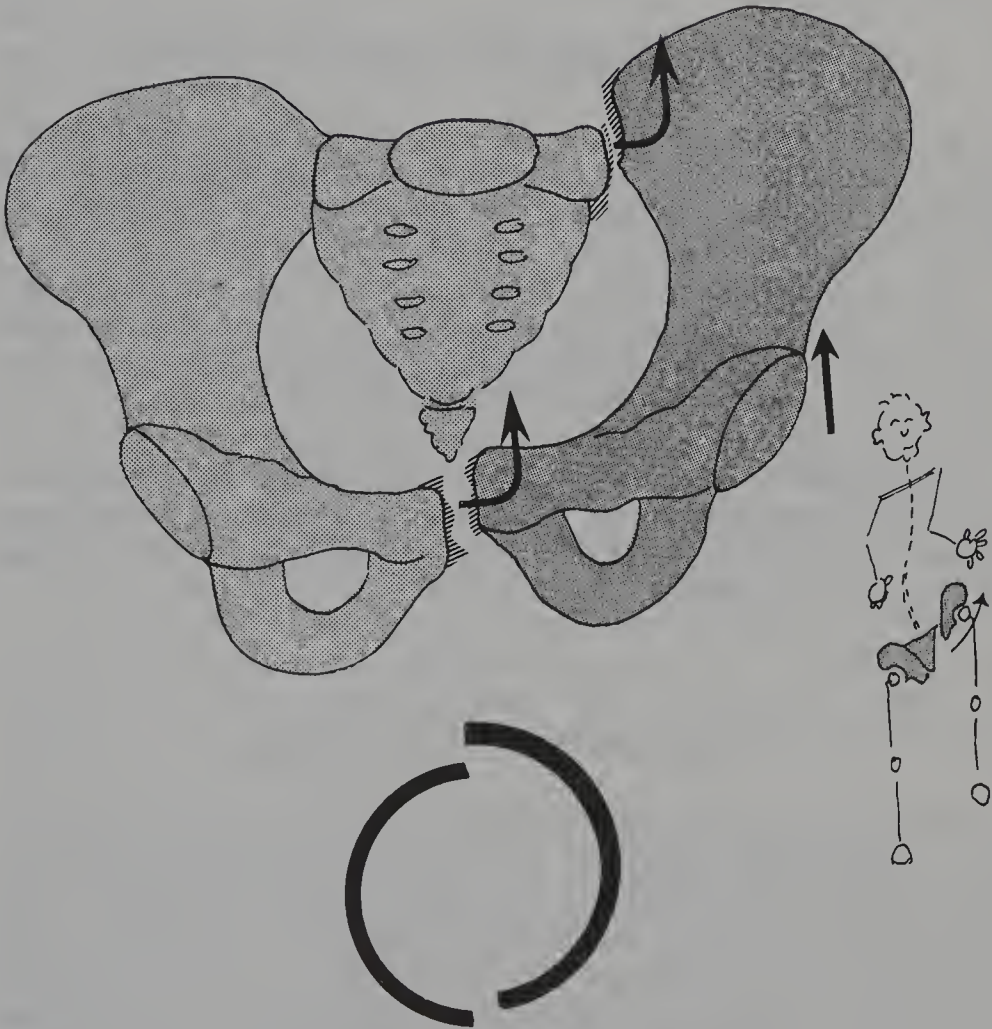


Figure 11-26. Combined sacroiliac and symphysis pubic separation. The condition D in Figure 11-25 is depicted in which a direct trauma separates half of the pelvis with separation of the symphysis pubis and the unilateral sacroiliac joint. The fractured segment moves superiorly and posteriorly causing the leg on that side to shorten.

reveal secondary sacroiliac pathology but a computerized axial tomography (CAT) scan or bone scan can be diagnostic. No pelvic fracture should be casually evaluated without suspecting the possibility of sacroiliac involvement.<sup>57</sup>

## DIABETIC LUMBAR RADICULOPATHY

Sciatica without disk herniation must be eliminated in a patient with known diabetes.<sup>58</sup> Neuropathy is a well-recognized complication of diabetes mellitus, occurring in approximately 10% of patients with diabetes<sup>59</sup> and usually is found in the lower extremities.

Pain presents with sudden onset, is usually severe, and may require narcotic medication for relief. Weakness of the proximal leg muscles is often observed as are decreased sensation and hyporeflexia or areflexia. Because of the proximal extremity involvement, it resembles a higher disk entrapment. Tension signs are usually absent.

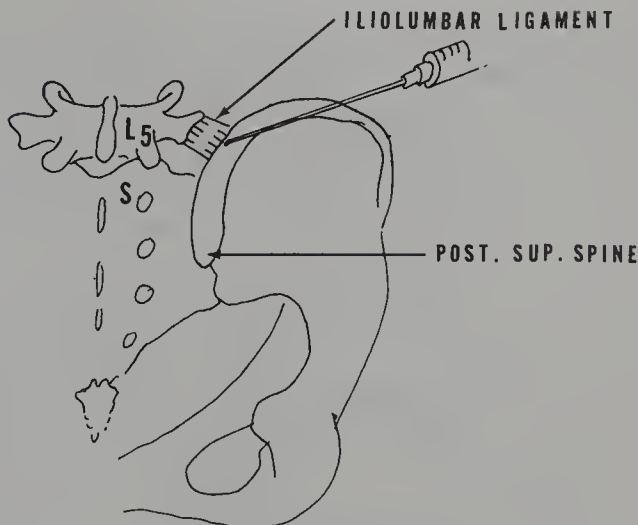
Spontaneous recovery may be expected,<sup>60</sup> with pain resolving in 16 to 20 weeks. Muscular strength recovery may be delayed for 1 year, and relapses are frequent (20%).<sup>61,62</sup> Diabetic control and conservative measures are indicated.

## ILIOLUMBAR SYNDROME

This syndrome has been considered a common cause of low back pain.<sup>63,64</sup> The patient suffers an acute onset of low back pain after an inappropriate movement or external trauma and then complains of pain in the posterior portion of the iliac crest. The patient usually can place one finger on the precise site of pain. The pain is usually severe and resolves into a constant localized ache aggravated by any low back movement but usually lateral flexion. It may come on after prolonged sitting or prolonged standing.

Characteristic pain occurs on lateral bending and one pelvic rim appears higher than the other. Forward bending is characteristically painless and unlimited. Pain on straight leg raising occurs in the precise area. Patrick's test also causes localized pain. The sacroiliac test (C in Fig. 11-22) reproduces pain in that area. The diagnosis is made by the above findings and is verified by localized tenderness relieved by an anesthetic infiltration into the ligament (Fig. 11-27).

**Figure 11-27.** Iliolumbar ligament. The iliolumbar ligament connects the transverse process of the fifth lumbar vertebra to the iliac crest. It can be palpated after localizing the posterior superior spine (post. sup. spine). Injection aims at striking the inner edge of the iliac crest at the fifth lumbar vertebral area.



## SACRALIZATION OF TRANSVERSE PROCESS

+

The transverse process of the fifth lumbar vertebra may have a congenital elongation that fuses or forms a pseudoarthrosis with the sacrum or the ilium. The usual site of this pseudoarthrosis is the fifth lumbar vertebra and the medial aspect of the adjacent ilium.

This condition is usually diagnosed radiologically on routine views for other pathology and is usually asymptomatic. If the fusion restricts motion, it may be responsible for excessive or inappropriate motion of cephalad vertebral functional units, causing pathological changes there that become sites of nociception.

Sciatic radiculopathy may result from sacralization when the nerve roots descending to form the sciatic nerve are encroached (Fig. 11-28). The condition is suspected when there is radiological evidence of its presence and the radiculopathy is caused by lateral flexion but not by forward flexion or extension. A CAT scan or magnetic resonance imaging (MRI) may be diagnostic, but confirmation may need relief of radicular pain from a localized injection of an anesthetic agent with or without steroids.

## SPINAL INFECTIONS

Spinal infections are rare, but they do occur and they can be ominous. Low back pain and tenderness, which accompany a systemic infection with septicemia, may indicate spinal infection.



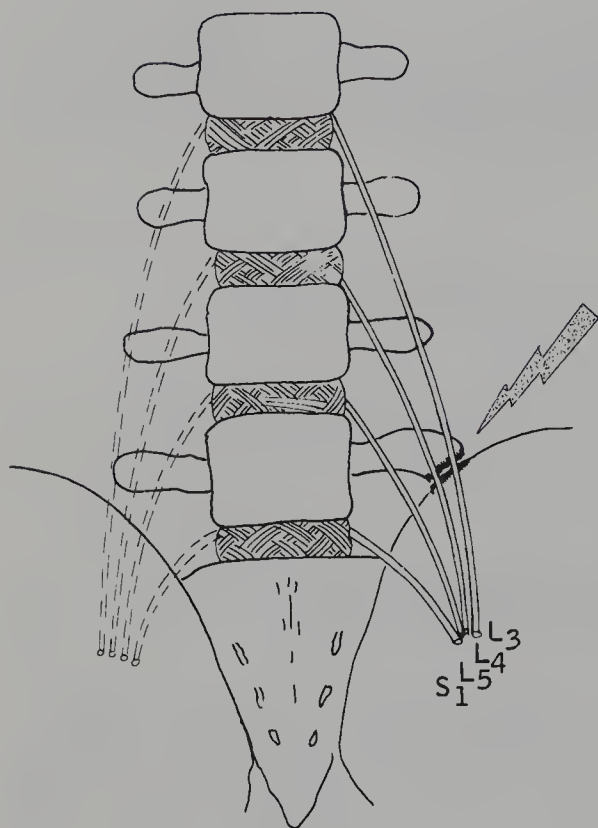


Figure 11-28. Sacralization of the transverse process. The elongated transverse process of the fifth lumbar vertebra has contacted the iliac crest and formed a pseudoarthrosis, which can cause low back pain. As the L3 to L4 nerve roots pass over the pseudoarthrosis they can be irritated resulting in referred pain in those dermatomes and paresis of the muscle innervated by these roots.

Osteomyelitis is an infection of a vertebral body resulting from hematogenous spread or spread from a local abscess into the vertebra. A frequent source of infection is a urinary tract infection, usually a staphylococcus infection. People with diabetes are prone to have this complication. The infected vertebral body may gradually undergo degeneration and destruction, with collapse and formation of a segmental scoliosis.

The condition is suspected from the onset of a nonspecific low back pain unrelated to any specific motion. Local tenderness can be elicited. The patient is toxic and undergoes a low-grade fever. X-rays are initially negative, but a blood count and elevated sedimentation rate are diagnostic. Blood cultures often become positive. A diagnostic bone scan has proved reliable. X-rays become positive within 4 to 6 weeks.

Treatment demands isolation of the specific bacterium followed by specific antibiotics. Supportive care is indicated, usually requiring a cast of the trunk to minimize scoliosis and deformity. Usually within 6 to 9 months the infection subsides and local fusion occurs, indicating the need for proper corrective immobilization of the segment during healing.

Discitis, a disk space infection, must also be suspected when the signs of local vertebral inflammation and systemic infection co-exist.



This type of infection occurs following a disk injection for diagnostic or therapeutic reasons but hematogenous spread is more frequent. This is currently more prevalent in people who are drug dependent and use the intravenous route for their addiction.

Pain is acute and excruciating. Local tenderness is prominent, as is severe muscle spasm as a guarding mechanism. All spinal movement is limited.

The sedimentation rate is markedly elevated, and bone scan becomes positive and diagnostic with the disk space becoming hazy and the endplates becoming disrupted and irregular. As the condition progresses, the disk degenerates and the endplates become destroyed, causing localized scoliosis, lordosis, or kyphosis. A needle biopsy becomes diagnostic and also delineates the specific bacterium involved.

Treatment is like that for osteomyelitis, with specific antibiotics, protective splinting or bracing, and ultimately the possibility of surgical evacuation followed by interbody fusion when the infection is controlled.

## TUMORS

Bone tumors, either primary or metastatic, must always be suspected when there is nocturnal pain, pain without precise movement aggravation, or pain that fails to respond to adequate conservative treatment.<sup>65,66</sup> Weight loss, cachexia, and debility frequently are present. The diagnosis is confirmed by proper evaluation of radiological, MRI, CAT, or myelographic tests.

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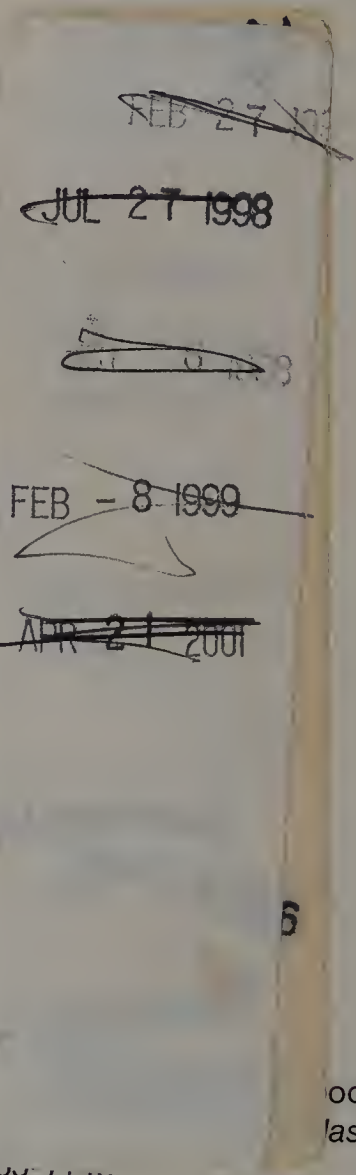
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